Medical
Taxon

The Journal of Pediatrics

VOL. 7 JULY, 1935 No. 1

Original Communications

PRESIDENT'S ADDRESS

THOMAS B. COOLEY, M.D.
DETROIT, MICH.

JOHN RUHRÄH

ONE year ago this chair was graced by the presence of one of the most distinguished of our fellows, for whom the presidency of the Academy was but one of many honors incident to a full life conspicuously well spent in his chosen field of endeavor. Today we grieve for his recent death.

John Ruhräh was in the truest sense a gentleman and a scholar. None could know him without perceiving his fine grain and the breadth and depth of his attainments. Essentially the wise practitioner, learned, experienced, and keen of insight, his influence extended beyond the walls of his consulting room, and he was a willing and valued counselor in the larger affairs of the community.

To many of us he was the beloved and honored friend, proved by long association. The yearly gatherings that refresh our spirits have been enriched by his cultured presence and illuminated by his genial, penetrating satire, sometimes phrased in clever, telling verse. He entertained and instructed us with the fruits of his research in pediatric history, in which he was the first authority. We were cheered and enlivened by his full-blooded zest of living. You of the younger generation who knew him less well must have admired the erudition of his writings and must have been inspired by his spirit when during the period of his presidency he visited a number of your cities in behalf of the young Academy.

All of us will long remember the stout heart that carried him bravely through a painful, crippling affliction and kept him with us in body and in spirit to his last days.

We have lost a whole man, whose memory will live long in our hearts, and whose name will go down in pediatric history. God rest him!

Delivered before the Fifth Annual Meeting of The American Academy of Pediatrics, New York, June 8, 1935.

THE PROGRESS OF THE ACADEMY

THE Academy of Pediatrics is now five years old. It has had rather surprisingly rapid growth in that period, and this seems to me a good time to try to appraise its development, and to inquire whether it is really fulfilling the high purposes which its founders had in mind for it, or whether it will prove after all to be just another medical society.

The story of its beginning has been told before and needs no repetition here. I should like, however, to call to your attention some phases in which pediatrics differs fundamentally from other special branches of medicine and which have been influential in making of this society an organization of a type quite unlike that of any other national society with which I am acquainted. It is commonplace enough to say that pediatrics deals rather with an age group than with special types of disease or special parts of the body; and that, because this age period is the one of growth and development, we are particularly concerned with the forestalling of disease and with securing for our clients the best possible development of their innate capacities, both physical and mental. Regarded in this broad view, it may almost be said that the alleviation of disease is incidental to modern pediatrics rather than its chief aim. Of no other special branch could any similar statement be made. Their range of effort is necessarily much narrower than ours, and the problems of their special organizations are less complicated.

Another peculiarity of pediatrics which has always seemed striking to me is that its practitioners constitute the most altruistic and idealistic group in medicine. I am sure that you will all realize this on consideration of the type of men and women whom one encounters at meetings such as this. I suppose the reason is that few who have not that outlook on life could go far in a specialty whose purposes are so essentially altruistic. These things have an important bearing on the course which the development of the Academy has followed.

The Academy was founded, not to promote the selfish interests of pediatrists, but to further the best development of the specialty, to foster proper relations with the many other agencies concerned with the problems with which we must deal, and to enable organized pediatrics to speak with a louder and more authoritative voice in all these various relations. To draw an analogy from another field, I think one might say that the founders' conception of the Academy corresponded to the idea of the craftsmen's guilds, as compared with the quite different spirit of trades-unionism which just now is prominent in medical circles.

To accomplish such purposes, the Academy must, of course, before all have a membership such as to be convincing of the solidarity of the specialty and the interest of its practitioners in the society's endeavors. It should, as nearly as possible, include in its ranks all competent pedi-

atrists in the country with interests any broader than their own private affairs. How near it has come to the attainment of this goal is realized by consideration of the present fellowship list, which now, after five years, includes probably two-thirds of the pediatrists in the United States who might be considered eligible. That their membership is more than a perfunctory gesture I think is demonstrated by the attendance and interest at this meeting.

Given an adequate organization, which I think we have, what has the Academy accomplished to justify itself?

Its work falls readily into three divisions: national, regional, and local, though these, of course, overlap and interlock.

The work of national scope is of necessity carried on by the Executive Board, the central office, and the various special committees, and is communicated to the general fellowship by reports submitted at these meetings and published in the JOURNAL. Such reports cannot of course give an accurate picture of all that goes on at any given time. The work of some of the special committees has been a direct follow-up of what was begun at the White House Conference; others have been busy with problems which have arisen since. A glance at the list of these committees will indicate how wide a field they cover. New ones are created from time to time as occasion arises.

With certain of their outstanding achievements I think that most of you are familiar. Perhaps the most convincing demonstration of the influence that the Academy can wield has been the great improvement in the position of pediatrics in the medical schools of the country, for which unquestionably the Academy's Committee on Medical Education, or at least its very competent chairman, is largely responsible. This committee is not resting on its oars, and we may look for further improvement in the status of pediatrics through its work and through the influence which the Academy can bring to bear upon the National Board of Medical Examiners. In line with the work of this committee has been the establishment and evident success of the Board of Pediatrics, for which the Academy can justly claim a large share of the credit.

The Committee on Hospitals and Dispensaries is carrying out what is really a monumental piece of work, whose results are being communicated to you through the JOURNAL as they are compiled. To some of you, not intimately concerned with hospital organization and development, these may seem only a mass of statistics, but they contain much-needed information and will be of service to every one in the hospital field.

A very important committee is that on Child Health Relations, which is doing notable work for development of proper relations between the Academy and the child health organizations, in particular, the departments of public health. The results of this work will show particularly in state and local fields, where there is much to be done.

The Committee on Clinical Investigation and Scientific Research has been studying some questions of general interest and importance. Since, at the time of writing this, I am not certain that their report will be ready for presentation at the meeting, I might be speaking out of turn in discussing it. I think I might safely mention a very appealing suggestion that I know the committee is prepared to make: that the Academy might undertake to promote coordinated research into matters which for the good of pediatrics especially need elucidation and that it might also announce a willingness to receive funds for research and allocate them to proper laboratories. There is at present no other organization in a position to do either of these things, whose potential value is, I think, quite evident.

My selection of examples of what some of the committees are doing is not at all to the disparagement of the others in the list, for I have reason to know that they are all active and doing their appointed tasks well. I have no doubt that, in creating them, selecting their personnel with care, and giving them the backing of an influential organization, the Academy is rendering an important service to pediatrics.

One could not omit from a summary of the accomplishments of the Academy in the national field some mention of the JOURNAL. I have reason to believe that all of you read and appreciate it, not merely as the official organ of the Academy, but as a journal of the first rank, which is meeting a real need, and has achieved a rapid and deserved success.

Before leaving this part of my subject, I should like to say a word about the central office. It is inevitable that an organization of this character should have a large proportion of its activities directed by an executive secretary from a central office. I suppose that few of you who have not been intimately concerned with the direction of the Academy have much conception of the volume of the routine business which is handled by a small office staff. At one time I had occasion to get from the Secretary some statistics on this point, with which I shall not bore you but which were rather surprising.

An important part of the work of the office has been to furnish proper representation for the Academy, and through it often for pediatrics, at important meetings, conferences with other organizations, legislative hearings, etc. This function falls naturally more often to the Secretary, though sometimes the President or the chairman of an appropriate committee may serve. As the strength and influence of the Academy increase, such calls upon it will doubtless come more frequently.

Speaking from necessarily intimate acquaintance, I am glad to be able to say that I believe the office to have functioned with remarkable efficiency and a minimum of friction or serious criticism and that I am not aware of any real errors of policy. I am particularly glad to realize that we have been able to avoid unfortunate entanglements in medical

politics and have not been vexed with futile discussions of "state medicine" and the other bogies of the day.

Doubtless the time will come, perhaps soon, when pediatrics, through the Academy, must make known its stand on some questions of general medical policy. When it does, I think we shall be strong enough to stand on our own well-considered conviction, with no question of dictation by any outside agency.

You are all familiar, I suppose, with what the Academy has done in your particular regions, but you have not all had occasion to get a bird's-eye view of the general success of the regional activities. It has been my pleasant duty during my term as your President to acquire some first-hand knowledge of the state of pediatrics and the part the Academy is playing in different sections, and I have had opportunity to hear comment from interested observers, such for instance, as members of the staffs of the Children's Bureau and the American Child Health Association. All of us have been impressed with the zeal and ability which the regional committees have shown in studying and attacking their own special problems and the striking results which they have achieved in stimulating pediatric effort and encouraging its extension into important fields previously more or less neglected. I hear testimony from many sources of an awakening of interest in public health affairs, of closer contact and better relations with important lay organizations, and of well-directed and effective effort to give pediatrics more of a part in county, state, and regional medical societies. This development has been especially apparent in the southern states, where the regional committee and some of the state chairmen have proved themselves notably efficient.

An interesting move in some districts is the institution under Academy auspices of courses in the fundamentals of pediatrics for general practitioners. This is not so subversive of our own selfish interests as it might seem on first thought. The greater part of pediatric practice will always be done by the general practitioner. We shall have our proper share if we do our work well, and it is no credit to us not to have the general standard as high as we can make it.

A surprisingly easy and successful development in the regional activities has been the merging with the Academy of various sectional societies. There was a good deal of doubt as to how nearly possible this would prove to be, and this phase was entered into with some hesitation. There seems to be little question, after the experiences of the last two years, that these same groups will come together with even more enthusiasm for regional meetings of the Academy than they had for their separate meetings.

One of the best services of the Academy has been to the individual practitioner, especially to the man in the community removed from the larger centers. To have a part in a national organization, working with

broad ideals for the general good, is a real stimulus. It adds to the sense of dignity in one's work, broadens one's knowledge, and adds to one's competence. I have no doubt that many a man is a better practitioner because of his fellowship in the Academy; that this is appreciated is shown by the keen interest manifest everywhere in all its doings.

I have said nothing so far regarding the scientific meetings, national and regional. These were not an essential part of the original scheme of the Academy but have been added, so to say, as a frosting for the cake.

The regional meetings last year all presented excellent programs and were enthusiastically attended. The national meetings have developed a rather unique method of mingling business with science, which has proved highly successful. I know of no meetings anywhere more instructive and entertaining than these. The Secretary has devoted a deal of thought and hard labor to their planning and successful conduct and has earned our gratitude.

I hope that I have succeeded in sketching for you a picture of the Academy as I see it—a lusty young society, already well in its stride of accomplishment and with a record of worthwhile achievement. I expect to see it grow in scope and extend its activities and to have and use to good advantage a very real power. I have no misgivings as to its future. It has had the fortune to be steered through its formative years by a group of the elder pediatrists who have worked together ever since child welfare attracted enough interest to become a "movement." They were founders of the Association for the Study and Prevention of Infant Mortality and of the American Child Health Association and took important part in the White House Conference; thus they were fitted by long apprenticeship to start the young Academy on the right path. Soon their places must be filled, one by one, from the next generation, and it is one of the present duties of the Academy, in which I think it will not fail, to develop the right men to be its leaders.

LEUCOCYTES IN INFANCY AND CHILDHOOD

A Statistical Analysis of 1,081 Total and Differential Counts from Birth to Fifteen Years

Katsuji Kato, M.D.
Chicago, Ill.

THIS study presents the nature of physiologic variations of total leucocytes and their differential counts from birth to fifteen years of age (here called "pediatric period"). Various aspects of such a study have appeared in the literature, but no single report gives a comprehensive leucocytic picture covering the entire pediatric period as here attempted. Studies most frequently quoted, such as made by Carstanjen (1900)[7] and by Schloss (1910),[31] are not only based on an insufficient number of observations but are even more inadequate in that they fail to give the total leucocyte counts for the age periods mentioned. Mere enumeration of percentage figures is meaningless. More important in evaluating normal and pathologic blood pictures is the absolute number of each cell type. Even reports in which total leucocyte counts are given with percentage figures (Gundobin,[13] Takasu,[36] Karnizki,[16] Perlin,[25] Rabinowitch,[26] Ockel,[24] Schüssler,[33] and Rominger[27]) are based on data too scant for reliable deductions. Attempts to construct hypothetical leucocytic curves on the basis of but a few counts, as ventured by Zibordi,[38] are always misleading.

The nearest approach to adequate studies of the white blood cells has been made in the neonatal period, such as done by Schiff,[30] Japha,[15] Scipiades,[29] Biffi and Galli,[6] McClanahan and Johnson,[21] Slawik,[34] Lucas and his associates,[20] Frank,[12] Arneth and Nienkemper,[3] Schmal, Schmidt and Serebrijski,[32] Forkner,[11] Stransky,[35] and Kovacs.[18] Though based on a comparatively limited number of observations, each contributes significant data defining the normal leucocytic picture of this period. Lippman[19] alone reports a complete satisfactory statistical analysis of the blood in newborn infants, but his data are limited to the first forty-eight hours of postnatal life.

The influence of various physical and chemical factors on the blood picture has been studied by numerous observers. Sunlight as well as ultraviolet rays have been used by Berner,[5] Thedering,[37] Sanford[28] and others, but with usually inconstant and even contradictory results. All the variations noted by these observers are well within the normal limits; therefore, their conclusions are not convincing. Likewise, the so-called digestion leucocytosis and leucopenia may be dismissed be-

From the Department of Pediatrics, University of Chicago.

cause published data give such indefinite results (Auricchio,[4] Hainiss and Heller,[14] Stransky,[35] Mitchell,[23] and Fletcher and Mitchell[10]). Washburn[40] has recently attempted a careful study of hourly and daily leucocytic variations in six young infants and demonstrated an absence of any fixed rhythm in the fluctuations, or of any definite correlation between such variations and physical or physiologic factors.

The present report analyzes 1,081 white cell estimations made in 1,037 cases, distributed as evenly as possible among thirty age periods from birth to fifteen years. Sixty-one of these estimations were counts repeated a second or third time on newborn infants at subsequent intervals. These counts were made on welfare clinic infants and children with nonhematologic conditions seen in the out-patient department of the Bobs Roberts Memorial Hospital for Children (1933-1934), and on newborn infants in the maternity wards of the Cook County Hospital (1931) and in the nurseries of the Chicago Lying-In Hospital (1934).

The total leucocyte counts used for this analysis fall within certain arbitrary range limits, varying with each age period, determined chiefly by the absence of pathologic conditions. The following range scale has thus been evolved:

Birth to 12 mo.	4,000-40,000
1- 2 yr.	6,000-13,000
3- 4 yr.	6,000-12,000
5-15 yr.	6,000-11,000

These counts were made with 2 per cent acetic acid as a diluent. Smears for the differential count were prepared by the slide and cover-glass method previously described,[17] and stained by the Pappenheim's panoptic combination technic (May-Grünwald stain, followed by a dilute Giemsa solution). The leucocytes were grouped into three main morphologic types: (1) granulocytes, (2) lymphocytes, and (3) monocytes.

The total granulocyte count includes the three recognized myeloid cells with polymorphous nuclei and specific granules: the neutrophilic, the eosinophilic and the basophilic granules. The leucoblasts, promyelocytes, myelocytes, and metamyelocytes, found in the blood of normal newborn infants, are also included in this group. (Since myeloblasts are practically never found in normal blood, even at birth, their classification needs no attention in the present study.) No myeloblasts were found in the smears studied.

The total lymphocyte count includes the three usual varieties, all morphologically identical, but designated according to their size as large, medium and small. Since plasma cells and Türk's irritation forms are so closely related to lymphocytes, they are thus classified,

but without affecting the general nature of the lymphocytic curve (because rarely found). No Rieder cells of lymphoid type were seen in the smears examined.

The total monocyte count includes the so-called large mononuclears, the transitional cells of the Ehrlich school, and the large monocytoid Rieder cells which were occasionally encountered. The clasmatocytes, the term first applied by Ranvier to the ameboid wandering cells of the common loose connective tissue, receive a separate classification in the hands of the workers using the supravital technic, but their appearance in the peripheral blood is still much debated. In the dried blood smears, these cells are not recognizable.

The data here presented have been treated statistically, giving the arithmetic mean or average values in absolute numbers for all groups of cells, together with the average percentage figures. The range of variations is shown by the maximum and minimum values for each age period. The standard variations (SD) from the arithmetic mean are also calculated, in absolute numbers, using the formula $\sigma = \sqrt{\frac{\Sigma m^2}{N} - M^2}$, which in turn are converted into percentage figures, the coefficient of variability (CV), using the formula, $CV = \frac{\sigma}{M} \times 100$.*

The infants and children, whose blood was examined for this study, fell into thirty consecutive age groups, as shown in Charts 1 and 2. During early infancy, particularly the period immediately following birth, the blood picture changes so rapidly and decisively that division into short intervals of time was considered necessary, whereas in older infants and children the periods are divided into yearly intervals. From these data, a graph, Chart 1, has been constructed showing the nature of age variations for each group of leucocytes, expressed in absolute numbers, in units of 1,000, together with a curve for the total number of the white blood cells.

The data here analyzed demonstrate that the total leucocyte count at birth averages 18,000, which by the end of the first twenty-four hours increases to over 22,000. The count then drops abruptly, reaching its lowest average level (from 8,000 to 9,000) on the fourth day. The lowest count in this study was 4,200 in a four-day-old infant. A similarly low average count of 4,400 was reported by McLean and Caffey[22] for infants ninety-six hours old. The number then increases slightly, up to 11,000, and this level is maintained until the third month. Thereafter, the curve gradually falls to about 7,500 at nine years, a level apparently maintained by normal children throughout pubescence and early adolescence.

The most reasonable of the many theories as to the cause of high leucocyte count at birth is the concentration theory based on the initial

*Complete data in tabular form, incorporating these calculations, are here omitted due to the lack of space but may be obtained from the author upon request.

weight decrease, largely due to dehydration. That this is not the only cause of neonatal leucocytosis, however, is evident since differential count reveals a correspondingly high percentage of the granulocytes and monocytes and a low percentage of lymphocytes. This granulocytosis with great increase in total count closely resembles the blood picture of acute infections in older children. Thus the neonatal leucocytosis may be interpreted, in part at least, to reflect rapid, temporary adjustment of the defense mechanism, upon sudden transition from sterile intrauterine life. However, the neonatal granulocytosis, both absolute and relative, drops precipitously, paralleling an equally rapid reduction in total count, reaching a low level of 4,470 (51.2 per cent) on the fourth day. This level is maintained with minor fluctuations for three weeks. Toward the end of the first month, the granulocytes drop further, reaching in the second month the lowest level of all periods (3,000, or 32.2 per cent). From here on, the total granulocytes gradually increase until the end of the pediatric period (from 5,000, or 62.6 per cent).

The granulocytic curve in Chart 1, of which neutrophiles naturally constitute the greater part, indicates fluctuations in total number. Eosinophiles and basophiles, in the newborn period, are comparatively high as to absolute numbers but throughout the remainder of childhood remain uniformly low both as to total number and percentage. Elder and Hutchinson[8] found no basophiles in the blood of newborns, but data prove their observations to be erroneous.

In the newborn period, there are a few morphologic peculiarities of granulocytes. Most immature in form is the leucoblast, characterized by its large size, rounded single nucleus with granular (occasionally finely reticular) chromatin pattern, frequently visible nucleoli, and abundance of coarse nonspecific azurophilic granules scattered throughout the cell. The leucoblast has often been referred to in the literature as an "unclassified" cell. But such a nomenclature is obviously the result of improper morphologic identification. Promyelocytes and myelocytes are rare, but metamyelocytes rather common during the first two weeks of postnatal life. The immature myeloid forms of the eosinophilic and basophilic series are practically never seen.

The behavior of the total number of lymphocytes is interesting. At birth the absolute number is slightly above 4,000 (but with a low percentage figure, from 20 to 25 per cent) which by the fourth day has moderately decreased to a low level of slightly under 3,000 (with a higher percentage figure, 31 per cent). From the second month on, the lymphocytic curve declines very gradually until the adult level (about 2,500) is reached at the age of fifteen. It is noteworthy that the lymphocytic and granulocytic curves cross at two age periods: (1) at the end of the first week when both cell types have identical absolute and relative figures (about 4,000, or 40 per cent), and (2) at

four years, when they again approximate one another (4,000, or 50 per cent). Although a few observers have commented upon this intersecting of the two curves, there seems to be no agreement as to the time of crossing. Lippman noted the approximation of the two cell types as early as the forty-eighth hour after birth, while Carstanjen and Schiff place the similarity at six and nine days, respectively.

The morphology of the neonatal period lymphocytes calls for comment. In this period the lymphocytes have been observed to be larger than those of later periods. Careful study of their neonatal nuclear

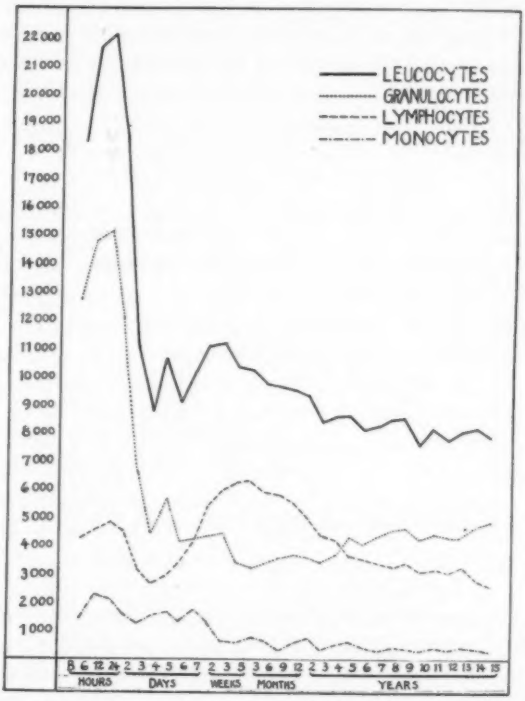


Fig. 1.—Curves of average total leucocyte counts from birth to fifteen years of age.

pattern often reveals a chromatin arrangement characteristic of immaturity. Frequently very finely and uniformly distributed, the chromatin granules are not definitely aggregated into heavy, coarse masses as seen in mature lymphocytes. The nucleoli are usually distinctly visible. The cytoplasm of these young lymphocytes is scant and stains rather heavily and often slightly granular. These differentiations may be viewed, regardless of cell size, as typifying immature lymphocytes or prolymphocytes, representing intermediate stage of lymphoid maturation between lymphoblasts and true mature lymphocytes.

The most interesting of all leucocytic variations lies in the monocytic curve. Monocytes in the newborn period are usually high, both in absolute count and percentage, varying the first two weeks from 1,340 to 2,200, and from 7.8 to 17.5 per cent per cubic millimeter. The third week produces a sharp drop to about 700 (6.2 per cent). During the following five or six months, the monocyte count averages about 600 (6 per cent), then drops slightly to an average of 500 (6 per cent), maintained until the sixth year. This constant average of 450 to 500, reached at the age of six years, continues throughout childhood.

Morphologically the neonatal monocytes also show evidence of immaturity. The nuclear chromatin of typically mature monocytes is definitely reticular, and the fine azure cytoplasmic dust, highly granular. But in immature monocytes, the nuclear chromatin is rather

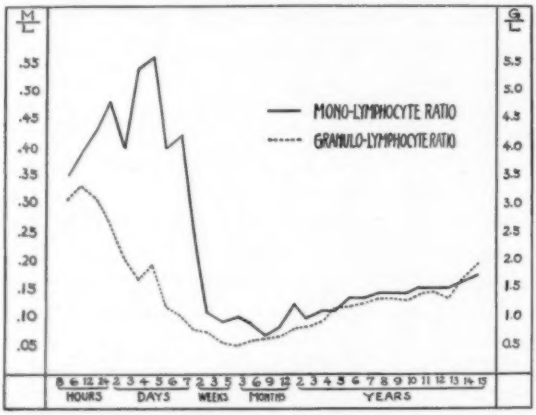


Fig. 2.—Curves of mono-lymphocyte and granulo-lymphocyte ratios.

granular, with distinctly visible nucleoli, and the cytoplasm less granular than that of mature cells. In these immature monocytes the azure dust is not as constantly marked as in older cells. Nuclei of the immature cell type may be smooth in contour, evincing little of the tendency toward deep indentations and fantastic configurations more typical of mature monocytes. These immature monocytes, or promonocytes, may represent the intermediate development between monoblasts and mature monocytes.

Interpretation of certain pathologic processes is based on the interrelationships of the three leucocytic cell types. Most important of these relationships is the monocyte-lymphocyte ratio (here called monolymphocytic ratio) obtained by dividing the absolute number of monocytes by the absolute lymphocyte number, and expressed by the formula $\frac{M}{L}$, but to evaluate abnormal conditions or fluctuations in

this ratio is misleading unless normal values are first definitely established. From the data here assembled a normal monolymphocytic ratio curve is constructed (Chart 2).

A similar relationship existing between total granulocytes and lymphocytes, designated as granulolymphocytic ratio, is calculated by division of total granulocytes by total lymphocytes, and expressed by the formula $\frac{G}{L}$ (also shown in Chart 2). The similarity of these two curves, except during the first week of postnatal life, is striking.

In certain conditions in which the monocytic or the reticuloendothelial system is stimulated, the monolymphocytic ratio is significantly altered. The monocytes usually increase, effecting a rise in the monolymphocytic ratio. If elevation of monocytes is at the expense of lymphocytes, then this ratio gives a still higher value. Clinically, the prognosis of tuberculous infection has been gauged according to the variation of the monolymphocytic ratio. A higher ratio value is said to prevail during the acutely active stage, while a lowering of the ratio denotes quiescence.

Likewise the granulolymphocytic ratio marks the progress of disease processes in which granulocytes are the determining factor. Usually the total granulocyte count is sufficient for clinical evaluation of pathology. But occasionally conditions are met with, in which only the relation between granulocytes and lymphocytes can clearly demonstrate the prognosis. Again, a parallelism existing between the monolymphocytic and the granulolymphocytic ratios in certain cases may further aid diagnosis and prognosis.

SUMMARY AND CONCLUSIONS

1. The normal variations of the leucocytes and their differential counts made on 1,037 individuals from birth to fifteen years of age, with a total of 1,081 estimations, are statistically analyzed.

2. The total leucocyte count is very high during the first two days, with a maximum height (average of 22,000) at the end of the first twenty-four hours. The drop in the count occurring on the third or fourth day is sudden and pronounced (average of 8,750). There is a slight temporary rise (up to averages of 10,000 to 11,000) during the first three months, after which the normal level (average of 8,000) is maintained, with a tendency, however, to a gradual fall, throughout the remaining period of infancy and childhood.

3. The total granulocytes, comprising neutrophiles, eosinophiles, and basophiles, are correspondingly high during the first three days of the neonatal period, falling sharply on the fourth day parallel with a similarly precipitous drop of the total leucocytes. From three weeks to four years, the total granulocytes are much lower in both relative and absolute numbers than the lymphocytes. The eosinophiles and

basophiles are constantly found throughout the pediatric period, but no significant variations are demonstrable.

4. The total lymphocytes are relatively low at birth and drop sharply on the third day, but they rise rapidly to reach their highest peak during the second month. From the third month on, the lymphocytes are greater in number than the granulocytes, until the fourth year when the two cell types are equally distributed. After the fifth year the lymphocytes steadily decrease until the fifteenth year when they number but half the granulocytes.

5. There is a marked monocytosis in the neonatal period, lasting throughout the first two weeks. Thereafter the absolute number of monocytes falls to a level more or less constantly maintained throughout the pediatric period.

6. There appears to exist a quantitative relationship between lymphocytes and monocytes, expressed as a ratio by dividing the percentage of monocytes by that of lymphocytes. This monolymphocytic ratio describes a standard curve of variations from infancy to childhood. A similar connection is also noted between lymphocytes and granulocytes (the granulolymphocytic ratio), calculated by dividing the percentage of total granulocytes by that of lymphocytes. These two ratio curves are found to be somewhat identical in nature, differing only in that the actual values of the granulolymphocytic ratio are approximately ten times greater than those of the monolymphocytic ratio. Clinical evaluation of certain pathologic conditions may be facilitated by the application of these ratios.

REFERENCES

1. Aitken, J.: J. Obst. & Gynaec. Brit. Emp. **1**: 414, 1902.
2. Arneth, J.: Arch. f. Gynäk. **74**: 145, 1905.
3. Arneth and Nienkemper: Ztschr. f. Kinderh. **34**: 263, 1923.
4. Auricchio, L.: Pediatria **28**: 1093, 1920; **29**: 977, 1921.
5. Berner, K.: (quoted by Thedering.[37])
6. Biffi, U., and Galli, P.: Riv. di clin. pediat. **6**: 1, 1908.
7. Carstanjen, M.: Jahrb. f. Kinderh. **52**: 215 and 333, 1900.
8. Elder, G., and Hutchinson, R.: Edinburgh M. J. **41**: 105, 1895.
9. Fischl, R.: Arch. f. Kinderh. **15**: 470, 1892.
10. Fletcher, E. G., and Mitchell, A. G.: Am. J. Dis. Child. **34**: 807, 1927.
11. Forkner, C. E.: Bull. Johns Hopkins Hosp. **45**: 75, 1929.
12. Frank, M.: Ztschr. f. Kinderh. **31**: 16, 1921.
13. Gundobin, N.: Jahrb. f. Kinderh. **35**: 187, 1893.
14. Hainiss, E., and Heller, S.: Monatschr. f. Kinderh. **27**: 512, 1924.
15. Japha, A.: Jahrb. f. Kinderh. **52**: 242, 1900; **53**: 179, 1901.
16. Karnizki, A.: Arch. f. Kinderh. **36**: 42, 1902.
17. Kato, K.: J. Lab. & Clin. Med. **18**: 527, 1933.
18. Kovacs, E.: Ztschr. f. Kinderh. **128**: 244, 1930.
19. Lippman, H. S.: Am. J. Dis. Child. **27**: 473, 1924.
20. Lucas, W. P.; Dearing, B. F.; Hoobler, H. R.; Cox, A.; Jones, M. R.; and Smyth, F. S.: Am. J. Dis. Child. **22**: 525, 1921.
21. McClanahan, H. M., and Johnson, A. A.: Arch. Ped. **33**: 757, 1916.
22. McLean, S., and Caffey, J.: Am. J. Dis. Child. **30**: 810, 1925.
23. Mitchell, A. G.: Am. J. Dis. Child. **9**: 358, 1915.
24. Ockel, G.: Arch. f. Kinderh. **75**: 40, 1925.
25. Perlin, A.: Jahrb. f. Kinderh. **58**: 549, 1903.
26. Rabinowitch, D.: Arch. f. Kinderh. **59**: 161, 1912.

27. Rominger, E.: Jahrb. f. Kinderh. **103**: 1, 1924.
28. Sanford, H. N.: Am. J. Dis. Child. **37**: 1187, 1929.
29. Scipiades, E.: Arch. f. Gynäk. **70**: 630, 1903.
30. Schiff, E.: Jahrb. f. Kinderh. **54**: 1 and 172, 1901.
31. Schloss, O. M.: Arch. Int. Med. **6**: 638, 1910.
32. Schmal, S.; Schmidt, W.; and Serebrijski, J.: Ztschr. f. Kinderh. **41**: 102 and 728, 1926.
33. Schüssler, E.: Jahrb. f. Kinderh. **104**: 87, 1924.
34. Slawik, E.: Ztschr. f. Kinderh. **25**: 212, 1920.
35. Stransky, E.: Ztschr. f. Kinderh. **41**: 562, 1926.
36. Takasu, K.: Arch. f. Kinderh. **39**: 346, 1904.
37. Thedering, F.: Das Quarzlicht und seine Anwendung in der Medizin, Berlin, 1921, Gerhard Stalling.
38. Zibordi, F.: Ematologia Infantile Normale e Patologica, 1925, Mailand.
39. Warfield, L. N.: Am. Med. **12**: 457, 1902.
40. Washburn, A. H.: Am. J. Dis. Child. **47**: 993, 1934.

920 East Fifty-Ninth Street

ERYSIPELAS IN CHILDREN UNDER TWO YEARS OF AGE

Clinical Study

Charles S. Culotta, M.D.
New Haven, Conn.

THIS report is concerned with a review of the cases of erysipelas in children under two years of age who were admitted to the New Haven Hospital and Dispensary during the years 1924 to 1934. During the period fifty-eight cases in this age group came under observation. An attempt will be made to present the essential data collected from a review of these cases.

Sex.—In this series of cases it was observed that the morbidity was the same in both sexes, but the mortality was twice as great in males as in females—of the fifteen children who died ten were males.

Predisposing Factors.—Epidemiologic studies in erysipelas have shown the contagiousness of the disease to be relatively low. Cushing[1] found the history of contact with another case unusual. Chabrol and Waitz[2] reported a family and an institutional outbreak of erysipelas, but these authors point out that such epidemics are considered rare. The history of a streptococcic infection in some member of a family in which erysipelas occurs is not unusual.* Investigation of the histories of our cases showed that predisposing factors, such as colds and sore throats, either in the patient or in some member of the family, were not unusual. Cushing also noted the frequency of throat infections, otitis media, and mastoiditis in his cases. In this series erysipelas was observed in two newborn infants within twenty-four hours after birth whose mothers had the disease at the time of delivery. Both of these proved to be fatal cases with septicemia. Previous injury to the skin due to trauma, operative procedures such as circumcision, or minor local infections was noted to be a predisposing cause in very few instances. In several cases no history of any predisposing illness was obtained.

Region.—For convenience the initial site of the lesion was tabulated with respect to three separate regions: the face, including the head and neck; the extremities; and the body, including the chest, abdomen, and genitals. Table I presents the frequency with which each was involved.

From the Department of Pediatrics, Yale University School of Medicine, and the Pediatric Service of the New Haven Hospital and Dispensary.

*Dr. Joseph Bunim, formerly of this department, observed in an epidemiologic study the frequent occurrence of scarlet fever or allied streptococcal infections in members of families in which erysipelas was present.

TABLE I

PRIMARY SITE OF LESION

REGION		NO. OF CASES	% MORBIDITY
Face		27	47
Extremities		18	31
Body	Genitals	7	22
	Abdomen	6	

The region most frequently involved in these cases was the face in twenty-seven patients, or 47 per cent. The extremities were involved next in frequency (18, or 31 per cent) and the body in 13, or 22 per cent of the cases. Cushing stated that above one year of age the facial, or adult type occurs in about 80 per cent of the cases of erysipelas; in 10 per cent the extremities are involved, and in the remaining 10 per cent, the body, the last group carrying the highest mortality. In Cushing's thirty-nine cases in infants under one year of age, thirty cases showed involvement of the body with a 60 per cent mortality, while the other nine cases showed involvement of the face with only one death. Unfortunately the exact ages of Cushing's patients are not given. It was found in our series that the highest mortality occurred in patients under two months of age, twelve deaths out of a total of fifteen. The cases under two months of age are tabulated with respect to the site of the primary lesion and mortality.

TABLE II

PRIMARY SITE OF LESION IN INFANTS UNDER TWO MONTHS AND THE ASSOCIATED MORTALITY

REGION		NO. OF CASES	DIED	% MORTALITY
Face		7	5	71
Body	Abdomen	3	3	100
	Genitals	3	3	
Extremities		2	1	50

Although the number of cases is small, the high mortality in all groups is striking, in the body type, especially. It would seem from these meager data that in infants the age factor is of primary importance rather than the original site of the region involved.

Blood Picture.—Moderate elevation of the white blood count was the rule in the majority of the nonfatal cases. The average white blood count was above 20,000 with a predominance of polymorphs. Several patients had white blood counts as high as 39,000 per cubic millimeter. A low white count was observed usually in the fatal cases; there was only one exception—a child one year of age with localized abscesses in both feet following erysipelas of the extremities was found to have a white count of 2,000.

Course.—Clinical observation has shown the course of erysipelas to be variable. Patients are known to recover spontaneously in a comparatively short time. Most authorities agree that the average duration is about two weeks. Bauer[3] in his series observed the course to last from six days to two weeks. The course of the disease in this series was also found to be quite variable. In the very young infants (neonatal group) the disease usually ran a rapid, fatal course, death occurring within two to six days after the onset of the illness. In the nonfatal cases the average duration was seven to ten days, complete recovery occurring within five days in a few cases. A long-drawn-out febrile course was observed in those patients who developed a purulent complication.

Complications.—The complications observed in this series are grouped in Table III.

TABLE III

COMPLICATIONS OBSERVED IN FIFTY-EIGHT CASES OF ERYSIPELAS UNDER TWO YEARS OF AGE

COMPLICATION	NO. OF CASES	DIED
Peritonitis	5	5
Abscesses	6	1
Bronchopneumonia	3	1
Otitis media	7	0
Adenitis	3	0
Empyema	1	0
Balanitis	1	0
Septicemia*	16	15

*Usual finding in all fatal cases.

The advent of sepsis and peritonitis proved to be exceedingly grave. The other complications were comparatively benign. Localization of the infection with abscess formation was observed in six cases with one death, this case having a blood stream invasion. Grasso[4] noted that abscess formation is a common complication in children having erysipelas of the extremities. The hemolytic streptococcus was the causative agent recovered from the abscesses; rapid recovery followed incision and drainage.

Mortality.—The high mortality of erysipelas in infancy is well known. Table IV presents the various mortality figures as reported by different authors.

TABLE IV

REPORTED MORTALITY OF ERYSIPELAS IN INFANTS UNDER TWO MONTHS OF AGE

AUTHOR	UNDER 1 MONTH		UNDER 2 MONTHS	
	NO. OF CASES	% DEATHS	NO. OF CASES	% DEATHS
Grasso[4]	16	94	not stated	57
Tarnier[4]	38	94		
Roger[4]	14	90		
Shaffer[5]	10	80		

The mortality in this series is tabulated in Table V.

TABLE V

MORTALITY IN PRESENT SERIES

AGE	NO. OF CASES	DIED	% MORTALITY
0 – 1 mo.	10	8	80
1 – 2 mo.	10	4	40
3 – 12 mo.	24	1	4
13 – 24 mo.	14	2	14

Table V shows that under one month the mortality is exceedingly high, somewhat comparable to the reported mortality in this age group. Twelve of the total fifteen deaths in fifty-eight cases occurred in infants under two months of age, a mortality of 60 per cent, whereas a mortality under 8 per cent occurred in infants aged three months and older—the approximate mortality rate as given by Cushing. The disease is dangerous for infants under two months old and especially so for those under one month old.

Analysis of the blood cultures reveals some interesting data from the standpoint of prognosis in a given case of the disease. This factor has been given little or no attention in the reported cases. It was observed that all fatal cases in this series had positive blood cultures for the hemolytic streptococcus. One patient yielded in addition a positive culture for staphylococcus which was later considered a contaminant. In the patients who recovered, the blood cultures were usually sterile with the exception of one: a child, one month old, with a negative blood culture on admission developed a positive blood culture with the appearance of a localizing abscess in one extremity. Following drainage of the abscess, successive blood cultures were sterile. In general, it can be stated that the appearance of a blood stream invasion in erysipelas is a bad prognostic sign.

Treatment.—No attempt will be made to discuss in detail the efficacy of the treatment employed in these cases. A comparative method of study of the value of any therapeutic measure by presenting temperature charts or number of days hospitalized is limited in its application and usually misleading because of the small number of cases observed. There are given in the literature on the treatment of erysipelas numerous optimistic claims for a variety of therapeutic agents, but an equal number of pessimistic reports can be found of instances in which the same therapeutic measures were employed.

In general, no startling results were observed following any one form of therapy. Between 1924 and 1928 antistreptococcus serum was often given usually in conjunction with transfusion of citrated whole blood, but in the later years repeated transfusions was the treatment of choice.

A complete cessation of spread of the erysipelas was not observed in these cases regardless of the form of therapy employed. The high mortality in infants less than two months old is quite striking, although these cases received intensive treatment. In evaluating any form of therapy for erysipelas, factors such as age and the presence of a blood stream invasion are obviously of great importance, although optimistic reports on various types of therapy fail to give these factors due consideration. Bauer points out that the minimal mortality rate for erysipelas is 8 per cent and that all methods of treatment have not reduced the mortality effectively in any important list of cases. Grasso, after reviewing over 3,600 cases of erysipelas in all ages which had been treated by diverse methods, concludes: "Whatever the cure, be it specific or nonspecific, general or local, chemical or mechanical, all fail."

On the basis of clinical observation it can be said that blood transfusion is an invaluable supportive measure in the treatment of erysipelas in young children, as it is for any of the other acute febrile diseases.

SUMMARY

1. In a review of fifty-eight cases of children less than two years old, it was observed that the greatest mortality occurred during the neonatal period.

2. Blood stream invasion was a characteristic feature of the fatal cases.

CONCLUSIONS

Age and the presence of septicemia are two important factors which influence the prognosis of erysipelas in children.

REFERENCES

1. Cushing, H. B.: Canad. M. A. J. **21**: 276, 1929.
2. Chabrol, E., and Waitz, R.: Bull. et mém. Soc. méd. d. hôp. de Paris **47**: 669, 1931.
3. Bauer, E. L.: Pennsylvania M. J. **35**: 613, 1932.
4. Grasso, R.: Policlinico (sez. prat.) **39**: 1645, 1932.
5. Shaffer, A. J., and Rothman, P. E.: Am. J. Dis. Child. **33**: 116, 1927.

THE ANTIRACHITIC PROPERTIES OF IRRADIATED EVAPORATED MILK FED TO NORMAL BABIES UNDER HOME CONDITIONS

ROBERT A. STRONG, M.D., EMILE F. NAEF, M.D., AND
INA M. HARPER, M.D.
NEW ORLEANS, LA.

TEN years have elapsed since Hess, Weinstock, and their associates,[1] and Steenbock and his coworkers[2] demonstrated that it was possible to endow various foods with antirachitic potency by irradiating them with the ultraviolet ray. The experiments which resulted in this discovery were inspired by earlier observations that sunlight was beneficial in preventing and sometimes curing rickets. The desirability of identifying the substance or substances in the body of the infant capable of receiving and utilizing the benefits of sunlight was but a natural sequence. As a lead in this direction, it was found that when certain vegetable oils were irradiated and added in amounts of 0.1 c.c. daily to a diet that was otherwise known to produce rickets in rats, these oils were capable of affording protection against rickets. Moreover, the antirachitic substance was always present in the nonsaponifiable fraction and lacking in the saponifiable fraction. As the main constituent of the nonsaponifiable fraction is phytosterol, the desirability of ascertaining if this substance could be activated was at once apparent, and it was found to be possible. Cholesterol, one of the lipoid components of animal cells that corresponds to phytosterol in the plant cell, was next found to be capable of being activated with the ultraviolet ray. This in turn suggested that cholesterol and other sterols, which are now known to be present in human skin, were probably the substances which are activated by the ultraviolet ray of sunshine and consequently are capable of synthesizing the vitamin D in the body, thus preventing and curing rickets. To prove this, human skin and calf skin were subjected to irradiation with the ultraviolet ray and fed daily in 1 gm. per capita amounts to rats receiving a ricket-producing diet of low phosphorus content. A control series received nonirradiated skin. The rats receiving irradiated skin were regularly protected from rickets, and the control animals developed rickets. This justified the earlier belief that the beneficial effect of solar rays or artificial irradiation with the ultraviolet ray was due in some measure to the activation of the sterols which are so widely distributed in the skin. The successful irradiation of various foods followed soon

From the Department of Pediatrics, School of Medicine, Tulane University of Louisiana.

after, and Alfred Hess and Lewis[3] eventually demonstrated that when whole cow's milk was thus enriched with 50 Steenbock units of vitamin D to the quart, children receiving this milk were protected from rickets even when they received only enough to provide from 35 to 42 units in the twenty-four-hour intake. As a result, irradiated whole cow's milk has been made available in a number of localities, but its distribution as yet is not widespread and is restricted mostly to large cities. Recently, however, the irradiation by the Steenbock process of evaporated milk has added this advantage to an inexpensive type of milk, which is almost universally distributed and which has been rapidly growing in popularity since Marriott and Schoenthal[4] and Brennemann[5] invited attention, five years ago, to its superiority for infant feeding over other forms of milk. It seemed reasonable to suppose that the activation of evaporated milk with the vitamin D in this manner, would impart to it the same antirachitic potency which Hess previously showed irradiated cow's milk possessed, but a controlled study to determine if this really were true seemed to be desirable. So this clinical investigation was undertaken to determine the antirachitic properties of evaporated milk, the vitamin D potency of which had been enriched to 3.8 Steenbock units per ounce by irradiating it with ultraviolet rays from a carbon arc lamp in accordance with the process administered by the Wisconsin Alumni Research Foundation.

This investigation was begun in the latter part of December, 1933, and continued until the latter part of November, 1934, during which time observations were made on twenty-two infants fed on irradiated evaporated milk with added carbohydrate in conventional amounts. The infants were those which ordinarily are classified as "normal" or "well" infants. Most of them were selected at random from patients brought to the Newborn Clinic of the Department of Pediatrics of the Hutchinson Memorial Clinic for routine newborn examination and feeding directions; but a few were patients of other clinics under the control of the Department; some were private patients. Some discrimination was used in choosing mothers who were sufficiently intelligent to carry out instructions specifically and who were willing to cooperate fully for the entire period of the experiment. None of the babies received any vitamin D from any other source, nor did they receive any vitamin A other than that contained in the evaporated milk. All of them received vitamin C beginning about the fourth to the sixth week, and for the most part orange juice was used as a source of this factor. Tomato juice was used occasionally. No special selection of cases was made, with the exception that every effort was made to exclude premature infants, who are known to be extremely susceptible to rickets. One, however, was included in the series because the mother was so cooperative that it seemed desirable to include this baby to ascertain if he would be pro-

tected in spite of his physical handicap. An additional reason for the exclusion of premature infants was that it was desired especially to determine the value of irradiated evaporated milk in preventing clinical rickets in normal babies fed under home conditions. Institutional babies were excluded for the same reason.

As far as possible, various races and nationalities were included, and, because of known susceptibility to rickets, a few Italians and one negro formed a part of the group. Most of the babies, particularly in the irradiated evaporated milk group, were started between the ages of four and eight weeks, as it is in this age group that the opportunity to prevent rickets is most favorable. The babies were under observation until they were at least six months of age, and in a number of instances longer. To facilitate tabulating and recording the observations on each infant, a special folder containing five printed forms was prepared. These tabulations included the criteria to reveal the existence of clinical rickets, a careful history of the infants, the milk mixture, the amounts taken or rejected by the infant, notes on growth and development manifested by weight and measurement, and other data which it was believed might be helpful in formulating conclusions. The criteria to determine the presence or absence of clinical rickets were: beading, epiphyseal enlargement, bowing of the long bones, flaring of the ribs, craniotabes, bosses, and muscle tone. These were checked by roentgenograms. Record was kept of any illnesses, infections, character of sleep, and physical activities. In view of the fact that Jeans and Stearns[6] have so recently proved that infants fed on nonirradiated evaporated milk showed high retentions of nitrogen, calcium, and phosphorus, it was felt that a repetition of these determinations was unnecessary. Furthermore, it is becoming quite generally recognized that a parallelism between the concentration of blood phosphorus, bone ash, and the degree of rickets is by no means constant. Laurens[7] refers to this discrepancy by saying that "this paradoxical phenomenon—a rise of inorganic phosphorus unaccompanied by a healing of the epiphyses—shows that, although rickets usually is associated with a decrease of the inorganic phosphorus of the blood, this diminution cannot be regarded as an essential or inherent feature in the pathogenesis or the healing of this disorder. In addition to the systemic disturbance which is characteristic of rickets, there is probably a local disturbance at the epiphyses which prevents the anchorage of calcium and phosphorus in the cartilage and in the bones." Moreover, the object of the study was to ascertain whether or not clinical or roentgenologic rickets could be detected in a group of normal babies fed with irradiated evaporated milk under normal home conditions.

The study was made for the most part in the cooler months of the year, so little opportunity was afforded to give the babies sunbaths.

The only exposure to the sun was that which a baby might receive in this climate during the winter months, when he would be brought to and from the clinic fully clothed, and in the spring months, toward the close of the period of observation. It is, however, especially interesting to note that in New Orleans the months of June and July, 1934, were extremely cloudy. In June, for instance, there were 5.24 inches of rainfall, eighteen partly cloudy days, and four cloudy days; and in July there were 7.72 inches of rain, seventeen partly cloudy days, and nine cloudy days. These observations were recorded by Professor Laurens, of the Department of Physiology of Tulane University, who, for a number of years, has been measuring the total energy and distribution of sun and sky radiation in gram calories per square cubic centimeter per minute and in per cent of total, on a horizontal surface with an Eppley pyrheliometer. In these two particular months he was unable to take readings. It was felt, therefore, that ultraviolet radiation from the sunlight could not be regarded as an additional antirachitic factor of any great importance.

In short, the observations were made on infants living in the home under normal conditions and habits because of the fact that the most important indication seemed to be to determine the value of irradiated evaporated milk in the type of infant which really constitutes the vast majority among whom such a milk is used.

For the purpose of comparison, a second, but smaller, group of infants was fed on simple mixtures of whole cow's milk (boiled), water, and carbohydrate sufficient to supply their nutritional requirements. This control group received from 10 to 15 drops of viosterol daily as a source of vitamin D, and orange or tomato juice as a source of vitamin C. As in the first group, no additional vitamin A was supplied other than that contained in the cow's milk.

The formulas of the infants in both groups were calculated to provide an adequate amount of the necessary food elements, other than the vitamins. All the infants were given cereals between the fourth and fifth months, and vegetable purées were begun about the sixth month. Examinations were made at weekly intervals for about the first month of observation, and thereafter the intervals varied from one to three weeks. Tables I and II summarize the recorded observations, and contain all data of outstanding importance and interest. The dates selected are at approximately monthly intervals. Variations from this were made, however, so as to include important findings.

Tables I and II show the age, weight, any clinical findings indicative of rickets on the first and succeeding examinations, with notations of illnesses and results of roentgenograms of the forearms at the approximate age of six months, in the experimental and the control groups, respectively.

All the infants in both groups showed normal, or more than normal, gain in length and weight. Infections were about as frequent as might be expected in those infants living in the crowded, sometimes indifferently heated small homes.

Nine infants in the evaporated milk group and four in the control group showed at some time during the period of observation clinical evidences suggestive of rickets, as indicated in the tables. The muscle tone in all the babies, as determined by palpation, activity, and ability to sit alone, was normal at the conclusion of the study, and the suggestive evidence was no longer detectable.

Roentgenograms were taken of the forearms of each infant, at approximately the sixth month, to determine the existence of roentgenologic evidence of rickets at the completion of the study. None of the control group showed such evidence of rickets, while two of the evaporated milk group showed "slight evidence of rickets." One of these (J. E.) was not included in the study until he was fifteen weeks old and had received no vitamin D before that time. This child consumed a rather small amount of milk, 12½ ounces at the age of six and one-half months, which consequently gave him a maximum intake of 55.5 units of vitamin D. The second child (A. M.S.), observed from the eighteenth day of life, took a normal or rather large amount of milk for her weight, did not gain weight unusually fast, had only transient beading of the ribs, and sat alone at the age of four months. Nevertheless, at the age of five and one-quarter months, roentgenologically there was slight evidence of rickets.

In common with others, we feel that the roentgenologic evidence of rickets is more reliable than clinical signs. As has been pointed out by Eliot and Powers[8] and by Wilson,[9] clinical evidence obtained at a physical examination is based largely on a subjective estimate that permits a wide range of error. On the other hand, the condition of the bones as shown by a roentgenogram, offers an opportunity to interpret changes more satisfactorily.

The growth and general development of the infants on evaporated milk was above the average. This is believed to be due in part to the careful supervision given them and to the interest and cooperation of the mothers. Two of the group (M. L. and R. E. P.) made remarkable progress as soon as they were placed on it. All of the advantages of evaporated milk, which are now widely recognized, were apparent in this study.

The average number of vitamin D units taken daily, as computed from the average amount of irradiated evaporated milk taken by different age groups, is shown in Table III.

It will be seen that the unitage varied between 26.6 and 58.9 from the first week to the twenty-ninth week. This parallels almost exactly the

TABLE I

OBSERVATIONS MADE ON INFANTS FED IRRADIATED EVAPORATED MILK

NAME	DATE	AGE IN MO.	WEIGHT IN LB. AND OZ.	BEADING	EPIPHYSEAL ENLARGEMENT	BOWING	CRANIOTABES	BOSSES	MUSCLE TONE	ROENTGENOGRAM	REMARKS
J. M. B.	2/23/34	1½	9 4	–	–	–	–	–			
	3/23/34	2½	10 10	–	–	–	–	–			4/20/34 Common cold
	5/ 4/34	4	13 3	–	–	–	–	–			7/20/34 Common cold
	6/ 8/34	5	15 1	+	–	–	–	–			
	7/20/34	6½	16 2½	+	–	–	–	–	Sits	Negative	Negro
J. M. B.	1/17/34	2	9 11	–	–	–	–	–			
	2/16/34	3	10 14	–	–	–	–	–			2/16/34 Common cold
	3/16/34	4	11 11	–	–	–	–	–			
	4/23/34	5½	13 2½	–	–	–	–	–			
	5/21/34	6½	13 12	–	–	–	–	–	Sits	Negative	French
S. C. B.	1/12/34	2½	9 5	–	–	–	–	–	Good		3/9/34 Common cold
	2/ 2/34	3½	10 3	–	–	–	–	–	Good		
	3/ 2/34	4½	11 9	–	–	–	–	–	Sits		Italian deserted
	4/ 6/34	5½	12 6	–	–	–	–	–	Sits		
E. F. B.	5/ 5/34	1	4 14	–	–	–	+	–	–1		
	6/ 2/34	1¾	6 13	–	–	–	–	–	Normal		
	7/ 9/34	3	10 ½						Good		
	8/13/34	4	12 10½						Good		
	10/29/34	6⅞	15 5¼						Good	Negative	Premature twin

TABLE I—CONT'D

L. L. B.	3/19/34	½	9		–	–	–	–	–	Good		5/19/34 Common cold
	4/ 4/34	1	9	4	–	–	–	–	–	Good		July 1-30. In another city—had diarrhea; protein milk, two weeks, no vitamin D
	5/ 3/34	2	11	½	–	–	–	–	–	Good		
	6/ 6/34	3	11	15½	–	–	–	–	–	Good		
	7/30/34	4¾	12	10	–	–	–	–	–	Good		July 30—August 27. Irradiated evaporated milk (acidified)
	8/15/34	5½	13	12	–	–	–	–	–	Good		
	8/27/34	5¾	15	½	–	–	–	–	–	Good	Negative	
C. E. C.	4/25/34	2	9	10½	–	–	–	–	P	Fair		6/18/34 Brother developed diphtheria, and a culture from throat of this child was positive although no clinical evidence developed other than an unexplained temperature for three weeks
	5/21/34	3	11	6½	–	–	–	–	P	Fair		
	6/20/34	4	10	8	–	–	–	–	P	Fair		
	7/16/34	5	12	6½	–	–	–	–	P	Fair		
	8/20/34	6	14	3	+	–	–	–	P	Fair	Negative	
	11/ 2/34	8¼	17	14½	+	–	–	–	P	Sits	Negative	
G. C.	2/27/34	1	10	2	–	–	–	–	–			
	3/21/34	2	12	10	–	–	–	–	–			
	4/24/34	3	15	12	–	–	–	–	–			
	5/ 7/34	3½			–	–	–	–	–			
	5/16/34	4	16	14	–	–	–	–	–	Good		
	6/27/34	5	18	3	–	–	–	–	–	Sits	Negative	
C. D.	1/22/34	1¾	9	6½	–	–	–	–	P+	Normal		2/16/34 Common cold
	2/23/34	2¾	11		–	–	–	–	P+			4/ 5/34 Otitis media, acute, non-suppurative
	3/27/34	3¾	12	12	–	–	–	–	P+			
	4/24/34	4¾	14	3½	–	–	–	–	P+			5/30/34 Bronchitis, acute
	5/30/34	6	16	3½	+	–	–	–	P+			6/22/34 Lymphadenitis of cervical lymph nodes
	6/ 8/34	6½	16	7	+	–	–	–	P+		Negative	
J. E.	1/18/34	3¾	13	8	–	–	–	+++	–	Good		
	2/15/34	4½	14	8	–	–	–	+++	–	Good		
	3/10/34	5	15	3	–	–	–	+++	–	Good		
	3/31/34	5¾	16	1	+–	–	–	–	–	Good		
	4/27/34	6½									Slight evidence of rickets	

TABLE 1—CONT'D

NAME	DATE	AGE IN MO.	WEIGHT IN LB. AND OZ.	BEADING	EPIPHYSEAL ENLARGEMENT	BOWING	CRANIOTABES	BOSSES	MUSCLE TONE	ROENTGENOGRAM	REMARKS
B. C. F.	1/22/34	2	10 1½	–	–	–	–	–	Good		
	2/22/34	3	11 12¾	–	–	–	–	–	Good		
	3/22/34	4	13 3	–	–	–	–	–	Good		
	4/19/34	4¾	14 6½	–	–	–	–	–	Good		5/3/34 Common cold
	5/17/34	5¾	15 4	–	–	–	–	–	Good		
	5/29/34	6	15 11	–	–	–	–	–	Good	Negative	Italian
D. A. F.	2/ 2/34	2	10 9	–	–	–	–	–	Good		10D cod liver oil (40 drops) one week prior to start
	3/ 2/34	3	12 6½	–	–	–	–	–	Good		
	4/12/34	4½	14 3	–	–	–	–	–	Good		1/29/34 Common cold
	5/17/34	5½	15 12½	–	–	–	–	–	Good		2/10/34 Diarrhea for two days
	5/28/34	5⅔									5/17/34 Common cold
	6/13/34	6½	15 13½	–	–	–	–	–	Good	Negative	6/ 1/34 Measles
N. G.	1/ 8/34	⅓	8 5	–	–	–	–	–	Good		1/13/34 Common cold
	2/ 5/34	1⅓	10 6¾	–	–	–	–	–	Good		4/10/34 Common cold
	3/ 7/34	2⅓	12 9	–	–	–	–	P and F	Good		4/13/34 Acute bronchitis
	4/ 6/34	3⅓	14 12	–	–	–	–	P and F	Good		6/ 1/34 Common cold
	5/ 4/34	4⅓	15 1	–	–	–	–	P and F	Sits		
	6/ 1/34	5	15 13	–	–	–	–	P and F	Sits		
	6/15/34	5½	15 13	–	–	–	–	P and F	Sits	Negative	Italian
E. H.	2/ 3/34	2¾	10 1	–	–	–	++	–	Poor	Roentgenogram	
	3/ 9/34	4	11 12	–	–	–	++	–	Improved	was not made in	
	4/13/34	5	14 12	–	–	–	+	–	Good	this case because	
	5/22/34	6⅓	17	–	–	–	+–	–	Good	of unexpected breaking of the contact with this baby	No evidence of clinical rickets

TABLE I—CONT'D

G. A. LaB.	1/20/34	1½	9		–	–	–	–	–	Normal		
	2/ 3/34	2	9	15	–	–	–	–	–	Normal		
	3/10/34	3¼	12	6	–	–	–	–	–	Normal		
	4/ 7/34	4	13	15	–	–	–	–	–	Normal		
	5/ 5/34	5	15	2	–	–	–	–	–	Sits		
	6/ 2/34	6	15	14	–	–	–	–	–	Sits	Negative	
M. L.	2/ 2/34	3½	9	14	–	–	–	–	–	–2		
	2/22/34	4	11	2½	–	–	–	–	–	–1		
	3/22/34	5	13	10	–	–	–	–	–	–1		3/15/34 Common cold
	4/25/34	6	15	12	–	–	–	–	–	–1		6/13/34 Acute rhinitis
	5/30/34	7	17	6½	Slight	–	–	–	–	Good	Negative	
P. J. LeB.	2/23/34	½	7	3	–	– –	+	+ –	–	Poor		
	3/23/34	1½	8	15	–	–	+	+ –	–	Poor		
	6/15/34	4	14	2	–	–	+ –	–	–	Good		
	6/28/34	4½			–	–	–	–	–	Good	Negative	
M. L.	1/26/34	1¼	9	6	–	–	–	–	Slight	Good		
	2/16/34	2	10	12½	–	–	–	–	Slight	Good		2/ 6/34 Common cold
	3/28/34	3¼	12	15	–	–	–	–	Slight	Good		5/ 3/34 Common cold
	4/19/34	4	14	4	–	–	–	–	Slight	Good		5/22/34 Acute suppurative otitis media
	5/29/34	5¼	16	1	–	–	–	–	Slight	Good		6/13/34 Common cold
	6/27/34	6¼	16	8	+	–	–	–	Slight	Sits	Negative	
R. M.	1/ 4/34	2⅓	11	5	–	–	–	–	–	Good		
	2/ 6/34	3⅓	12	12¼	–	–	–	–	–	Good		
	3/ 8/34	4⅓	14	12	–	–	–	–	–	Good		3/ 1/34 Common cold
	4/19/34	5⅔	16	2	–	–	–	–	–	Good		
	5/19/34	6⅔	16	11	–	–	–	–	–	Sits		
	6/15/34	7⅔	17	4	–	–	–	–	–	Sits	Negative	

TABLE I—CONT'D

NAME	DATE	AGE IN MO.	WEIGHT IN LB. AND OZ.	BEADING	EPIPHYSEAL ENLARGEMENT	BOWING	CRANIOTABES	BOSSES	MUSCLE TONE	ROENTGENOGRAM	REMARKS
R. E. P.	2/16/34	2½	7 12	–	–	–	–	–	Fair		Received cod liver oil 1½ mo. prior to start
	3/14/34	3½	10 2½	–	–	–	–	–	Fair		
	4/11/34	4½	11 9	–	–	–	–	–	Good		3/ 7/34 Acute suppurative otitis media
	5/11/34	5½	13 6	–	–	–	–	–	Good		3/14/34 Common cold
	6/ 8/34	6½	15 9½	–	–	–	–	–	Sits	Negative	5/ 4/34 Common cold 5/11/34 Fracture of left clavicle 5/25/34 Common cold
A. M. S.	1/20/34	⅜	7 9	+	–	–	–	–	Normal		1/20/34 Thrush of mouth
	2/ 3/23	1	8 3	+	–	–	–	–	Normal		3/31/34 Common cold
	3/10/34	2½	10 10	–	–	–	–	–	Normal		5/ 2/34 Acute rhinitis
	4/ 7/34	3	12 2	–	–	–	–	–	Sits		
	5/ 5/34	4	13 3	–	–	–	–	–	Sits		
	6/ 2/34	5	14 13	–	–	–	–	–	Sits		
	6/ 8/34	5½		–						Slight evidence of rickets	
N. T.	2/23/34	4	10 6½	++	–	–	–	–	–1		
	3/24/34	5	11 10	+	–	–	–	–	Normal		3/10/34 Nasopharyngitis
	4/21/34	5¾	12 10	–	–	–	–	–	Normal		3/31/34 Nasopharyngitis
	4/28/34	6		–	–	–	–	–	Normal		
	5/19/34	6¾	13 10	–	–	–	–	–	Sits		
	6/ 2/34	7	13 12	–	–	–	–	–	Sits	Negative	
M. V.	3/ 2/34	¼									
	4/10/34	1½	11	–	–	+–	–	–	Good		
	5/25/34	3	13 9	–	–	+–	–	–	Good		5/17/34 Thrush of mouth
	6/18/34	3¾	14 12	–	–	+–	–	–	Good		
	6/28/34	4								Negative	

TABLE II

OBSERVATIONS MADE ON INFANTS FED WHOLE COW'S MILK

NAME	DATE	AGE IN MO.	WEIGHT IN LB. AND OZ.		BEADING	EPIPHYSEAL ENLARGEMENT	BOWING	CRANIOTABES	BOSSES	MUSCLE TONE	ROENTGENOGRAM	REMARKS
D. G. B.	3/13/34	4	14	11½	–	–	–	–	–	Good		
	4/12/34	5	17		–	–	–	–	–	Good		Had been fed S.M.A.
	5/ 3/34	5½	18	1	–	–	–	–	–	Sits		
	5/31/34	6½	18	13½	–	–	–	–	–	Sits	Negative	Hyperplasia of thymus
D. O. C.	3/23/34	2½	11	6½	–	–	–	–	–	Good		
	4/27/34	3½	12	4	–	–	–	–	–	Good		
	5/18/34	4½	13	3	–	–	–	–	–	Good		
	6/15/34	5½	14	3	–	–	–	–	–	Good		5/25/34 Common cold with slight diarrhea
	7/ 2/34	6	14	1	–	–	–	–	–	Sits	Negative	
R. E.	7/18/34	1¼	10	12	–	–	–	–	–	Flabby		Cod liver oil (4 teaspoonfuls daily) was given in this case instead of viosterol
	8/29/34	2½	13	14	–	–	–	–	–	Hard		
	9/12/34	3	14	10	–	–	–	–	–	Hard		
	10/10/34	4	15	14	–	–	–	–	–	Sits		
	11/ 2/34	4¾	18		–	+	–	–	–	Good	Negative	
A. F.	3/13/34	1	9	9	+	–	–	–	–	–		
	4/10/34	2	11	11	–	+	–	–	–	Flabby		4/10/34 Stomatitis
	5/15/34	3	14	11	–	+	–	–	–	Flabby		6/11/34 Acute nonsuppurative otitis media
	6/11/34	4	16	5	–	+	–	–	–	Hard		
	7/ 5/34	5	17	6	–	–						
	9/ 7/34	6				–					Negative	

TABLE II—CONT'D

NAME	DATE	AGE IN MO.	WEIGHT IN LB. AND OZ.	BEADING	EPIPHYSEAL ENLARGEMENT	BOWING	CRANIOTABES	BOSSES	MUSCLE TONE	ROENTGENOGRAM	REMARKS
M. M. H.	6/19/34	1½	10 5	–	–	–	–	–	–		Cod liver oil (4 teaspoonfuls daily) was given in this case instead of viosterol
	7/17/34	2½	11 10	–	–	–	–	–	–		
	8/21/34	3½	13 3	–	–	–	–	–	–		
	9/18/34	4½	14 8	–	–	–	–	–	Sits		
	10/31/34	6	16 4½	–	–	–	–	–	Firm	Negative	
J. P.	4/ 5/34	¾	6 5½	–	–	–	–	–	Fair		
	5/ 8/34	1¾	8 10½	–	–	–	–	–	Good		
	6/11/34	3	11 4	–	–	–	–	–	Good		
	7/ 9/34	4	12 13½	–	–	–	–	–	Good		
	7/23/34	4½	13 14½	–	–	–	–	–	Good		
	8/15/34	5	14 14	–	–	–	–	–	Good	Negative	Italian
J. A. W.	2/23/34	1¾	10 3½	–	–	–	–	–	Good		
	3/17/34	2¾	12 7½	–	–	–	–	–	Good		
	4/21/34	3¾	14 9	–	–	–	–	–	Good		
	5/19/34	4¾	16 2	–	–	–	–	–	Good		5/12/34 Fever, cause undetermined
	6/ 1/34	5	16 13	–	–	–	–	–	Good	Negative	
J. S.	2/20/34	1½	10	–	–	–	+	–	Hard		2/20/34 Common cold
	3/13/34	2	10 15	+	–	–	–	–	Hard		
	4/17/34	3	12 5	–	+	–	–	–	Hard		
	5/15/34	4	13 9	–	+	–	–	–	Hard		
	6/ 5/34	4¾	14 4½	–	+	–	–	–	Hard		
	7/13/34	6							Sits	Negative	

TABLE II—CONT'D

NAME	DATE	AGE IN MO.	WEIGHT IN LB. AND OZ.	BEADING	EPIPHYSEAL ENLARGEMENT	BOWING	CRANIO-TABES	BOSSES	MUSCLE TONE	ROENTGENO-GRAM	REMARKS
W. S.	3/15/34	1½	10 6½	–	–	–	–	–	–		
	4/17/34	2½	12 9½	–	–	–	–	–	–		
	5/15/34	3½	14 8½	–	–	–	–	–	Good		
	6/27/34	5	16 1	–	–	–	–	–	Good	Negative	6/13/34 Common cold
	7/11/34	5½	16 12½	–	–	–	–	–	Good		6/27/34 Lymphadenitis of cervical lymph nodes
S. S.	12/ 2/33	1½	10 6	–	–	–	–	–	Hard		12/2/33 Circumcised
	1/ 9/34	2	12 1	–	–	–	–	–	Hard		
	3/13/34	4	16 6	+	+	–	–	–	Soft		
	5/ 8/34	5¾	17 15	–	–	–	–	–	Hard		
	6/12/34	6	19 12	–	–	–	–	–	Hard		
	6/28/34	6½								Negative	
R. M. T.	5/18/34	1¾	9 12	–	–	–	–	–	Normal		Cod liver oil (3 teaspoonfuls daily) was given in this case instead of viosterol
	6/13/34	2¾	12 3	–	–	–	–	–	Normal		
	7/18/34	3¾	13	–	–	–	–	–	Normal		
	8/15/34	4¾	15 3	–	–	–	–	–	Normal		5/29/34 Diarrhea
	9/12/34	5¾	17	–	–	–	–	–	Normal		6/10/34 Pertussis
	10/10/34	6½	18 12	–	–	–	–	–	Normal		8/ 1/34 Common cold
	11/21/34	7	20	–	–	–	–	–	Normal	Negative	8/29/34 Impetigo contagiosa

number of units received daily by the youngest infants and the infants reaching the age of sixteen weeks and above, in the study recently completed by Jeans and Stearns,[10] in which the vitamin D was supplied from three different sources. These sources were irradiated evaporated milk, evaporated milk containing the nonsaponifiable fraction of cod liver oil (Zucker concentrate), and nonirradiated evaporated milk plus vitamin D given separately as cod liver oil. The diets were changed so that each infant received each diet in turn, and in none of these babies was there any evidence of clinical or roentgenologic rickets although one baby showed some evidence of decreasing bone density. This in turn seemed to parallel the observations as to unitage of Hess and his associates, to which previous reference has been made. In New Haven Wilson[9] recently studied thirty-three infants on milk fortified with the Zucker concentrate and observed a moderate degree of rickets in two of the group.

TABLE III

OUNCES OF IRRADIATED EVAPORATED MILK TAKEN DAILY BY 22 INFANTS

AGE PERIODS IN WK.	AVERAGE (OZ.)	RANGE (OZ.)	AVERAGE NUMBER UNITS VITAMIN D
1 – 4	7.0	6.0 – 10	26.60
5 – 8	8.7	6.0 – 12	33.06
9 – 13	10.6	7.5 – 15	40.28
14 – 17	12.4	8.5 – 16	47.12
18 – 21	14.3	10.0 – 16	54.34
22 – 25	15.0	12.5 – 16	57.00
26 – 29	15.5	15.0 – 16	58.90

COMMENT

For the purpose of comparing the incidence of rickets in the two groups of children in this study, there are available data in four studies made in the South. Ten years ago, DeBuys and von Meysenbug[11] reported a rachitic incidence of 34 per cent in a group of breast-fed infants in New Orleans. In 1928 C. T. Williams[12] reported that rickets in a mild form prevails to an extent of about 45 per cent in New Orleans children three years of age or less. In 1930 F. T. Mitchell,[13] Memphis, Tenn., reviewed the literature on the incidence of rickets in the southern part of the United States and reported his observations on 1,000 Memphis children, from four months to four years old. Five hundred of these were white, and 500 were colored. The incidence of rickets was 49.8 per cent in the white and 87.6 per cent in the colored. In the same year, Crawford and Williamson[14] reported that, in a group of infants studied in New Orleans, 42.9 per cent, who had received cod liver oil regularly from the tenth day of life on, showed roentgenologic evidence of rickets before the sixth month; and of those receiving cod liver oil irregularly, 69.2 per cent showed rickets by the sixth

month. In those who received no cod liver oil, the roentgen ray revealed rickets in 70 per cent. Again in 1934, C. T. Williams with the collaboration of Kastler,[15] in a study of sixty bottle-fed and twenty breast-fed New Orleans infants, observed from birth through the first six months of life, from May, 1931, to January, 1933, found clinical rickets in 35 per cent of the breast-fed and in 85 per cent of the bottle-fed infants and roentgenologic evidence of rickets in 90 per cent of the bottle-fed infants and 35 per cent of the breast-fed infants. All of these infants were given sunbaths outdoors routinely when the weather permitted and received cod liver oil fortified with viosterol in amounts which fully met conventional prophylactic dosage.

SUMMARY

1. A clinical investigation was undertaken to determine the antirachitic properties of evaporated milk, the vitamin D potency of which had been enriched to 3.8 Steenbock units per ounce by irradiating it with ultraviolet rays from a carbon arc lamp, in accordance with the process administered by the Wisconsin Alumni Research Foundation.

2. Twenty-two normal, artificially fed infants, living in poor to middle-class homes, were fed irradiated evaporated milk without any additional vitamin D, during a period of study extending from December, 1933, to November, 1934. This period embraced approximately the first six months of the life of the individual infants.

3. At the beginning of the study, five infants were between the ages of one and four weeks; nine were over four and under eight weeks; seven were over eight weeks and under thirteen weeks; and one was fifteen weeks of age. Two Italians and one negro child were included, and the remainder were of miscellaneous American parentage, the French strain being strong in the city. One premature infant was included.

4. A control group of eleven normal infants was fed whole cow's milk formulas, with from 10 to 15 drops of viosterol daily or 4 teaspoonfuls of plain cod liver oil as the source of vitamin D.

5. The average daily consumption of evaporated milk ranged from 6 ounces, in the one-to-four-week group, to 16 ounces in infants six months of age or over, giving a variation of 26.6 units to 58.9 units in the average vitamin D content of the food.

6. Roentgenograms of the forearms of the twenty-two infants fed irradiated evaporated milk, taken at approximately six months of age, showed "slight evidence of rickets" in two of the infants. This indicates an incidence of mild rickets in 9 per cent of this group. None of the infants in the control group showed roentgenologic evidence of rickets.

CONCLUSIONS

From the results obtained in this study, it seems reasonable to conclude that in irradiated evaporated milk containing 3.8 Steenbock units to the ounce, we have an inexpensive, pleasant-tasting, convenient source of vitamin D, which is almost universally available and which, when fed to the average normal infant in the average or below average home in this latitude, will protect about nine out of ten such infants from rickets. It thereby offers a source of vitamin D in an amount sufficient to reduce greatly the incidence or severity of rickets among patients whose parents either have not been educated to the necessity of antirachitic supplements to the food of all babies or who cannot afford them.

The impression is gained, however, that the irradiation of evaporated milk as yet should not be relied on exclusively to provide protection to all infants throughout the first year. It should be supplemented with additional vitamin D units from one of the conventional sources, particularly in the early months of life, when the intake of irradiated evaporated milk may be insufficient to provide the unitage necessary to protect the infant. Obviously, when irradiated evaporated milk is used, the necessary amount of additional antirachitic factors will be considerably less than when infants are fed a milk in which the vitamin D potency has not been thus enriched.

REFERENCES

1. Hess, A. F., and Weinstock, M.: Proc. Am. Pediat. Soc. (June 7), 1924; abstracted in Am. J. Dis. Child. **28**: 256, 1924; Proc. Soc. Exper. Biol. & Med. **12**: 5, 1924-25.
   Hess, A. F., Weinstock, M., and Helman, F. D.: Proc. Soc. Exper. Biol. & Med. **22**: 227, 1924-25; J. Biol. Chem. **62**: 301, 1924-25; **63**: 297, 305, 1925.
   Hess, A. F., and Weinstock, M.: J. Biol. Chem. **64**: 181, 1925.
2. Steenbock, H., and Black, A.: J. Biol. Chem. **61**: 405, 1924.
   Steenbock, H., and Nelson, M. T.: J. Biol. Chem. **62**: 209, 1924-25.
3. Hess, A. F., and Lewis, J. M.: J. A. M. A. **99**: 647, 1932.
4. Marriott, W. McKim, and Schoenthal, Ludwig: Arch. Pediat. **46**: 135, 1929.
5. Brennemann, Joseph: J. A. M. A. **92**: 364, 1929.
6. Jeans, Philip C., and Stearns, Genevieve: Am. J. Dis. Child. **46**: 69, 1933.
7. Laurens, Henry: The Physiological Effects of Radiant Energy, New York, 1933, The Chemical Catalog Company, Inc.
8. Eliot, Martha M., and Powers, Grover F.: J. A. M. A. **102**: 1823, 1934.
9. Wilson, William R.: J. A. M. A. **102**: 1824, 1934.
10. Jeans, Philip C., and Stearns, Genevieve: Proc. Soc. Exper. Biol. & Med. **31**: 1159, 1934.
11. DeBuys, L. R., and von Meysenbug, L.: J. A. M. A. **83**: 1563, 1924.
12. Williams, C. T.: Am. J. Dis. Child. **35**: 590, 1928.
13. Mitchell, F. Thomas: South. M. J. **23**: 228, 1930.
14. Crawford, Rena, and Williamson, G. Richarda: New Orleans M. & S. J. **83**: 219, 1930.
15. Williams, C. T., and Kastler, A. O.: J. Pediat. **4**: 454, 1934.

INTUSSUSCEPTION OCCURRING IN THE COURSE OF PARATYPHOID FEVER

JOSEPH MIGNONE, M.D., AND JAY M. ARENA, M.D.
DURHAM, N. C.

INTUSSUSCEPTION is a relatively rare complication of infantile diarrhea. Hess[1] reported two patients, one an eight-month-old child who had diarrhea for several days and in whom intussusception developed a few days after recovery and the other, a four-month-old child who had diarrhea for three days preceding the intussusception. Lichtenstein[2] reported 593 cases of intussusception, twenty-one of which followed diarrhea; Fitz[3] reported diarrhea lasting for more than ten days in thirteen of forty-five cases of intussusception; Perrin and Lindsay,[4] in a study of 400 cases of intussusception, stated that there was no relation between seasonal diarrhea and intussusception. However, in their series 11 of 156 cases of ileocecal intussusception, three of 126 ileocolic intussusception, and 6 of 19 colic intussusception had a history of preceding diarrhea. Moreton[5] reported one patient who had intussusception during the course of typhoid fever and ten in whom it developed during convalescence, but none of these patients had diarrhea. Six cases of intussusception during convalescence from typhoid fever were reported by Madelung[6] and Burhaneddin;[7] diarrhea was not mentioned. Holzmann[8] reported the occurrence of intussusception in a child on the fiftieth day of bacillary dysentery. The stools numbered from two to three daily and were of normal consistency for two weeks prior to the development of intussusception.

The three cases reported here are of infants in whom intussusception developed during the course of diarrhea; in two of them *B. paratyphosus* B was isolated from stool cultures.

CASE 1.—W. P., a white infant, thirteen months old, was admitted to the hospital July 18, 1934, because of diarrhea for seven days. The family and past history were irrelevant. The patient had been well until July 11, when suddenly a bloody, mucoid diarrhea developed, and he had as many as fifteen stools daily. He took his feedings well until July 16, when he began to vomit all that he ate. On July 17, he had only one small, bloody stool; and on July 18, the day of admission, he had two similar stools and had become comatose. The temperature on admission was 40.5° C. (105° F.); he was drowsy, dehydrated, and critically ill. His abdomen was distended and tympanitic, but no mass could be palpated externally or by rectal examination. When admitted at 4:00 P.M., the patient was given 200 c.c. 10 per cent glucose intravenously, and a continuous subcutaneous infusion of saline was

From the Department of Pediatrics, Duke University School of Medicine, and the Duke Hospital.

started. The temperature rose to 41.3° C. (106.3° F.), and an alcohol sponge was given. The CO_2 capacity of the blood was 73.6 volumes per cent, and the blood chlorides, 371 mg. per cent. At 10:30 P.M., he was given 130 c.c. 2.5 per cent saline intravenously. At 4:00 A.M. the next morning the temperature rose to 42° C. (107.6° F.), and at 8:00 A.M. it was 42.3° C. (108.3° F.). Each time an alcohol sponge was given, and the temperature dropped about one degree. He had not vomited since admission but had passed several small, bloody, mucoid stools, containing no fecal material. Two enemas were ineffectual. Although the patient was thought to have an intussusception, his condition was too poor for operation. He died at 3:00 A.M. on July 20. Stool cultures taken before death and at necropsy revealed no typhoid or dysentery organisms. At autopsy two intussusceptions, one of 50 cm. and the other of 10 cm., above the ileocecal valve were found. Sections of the small intestine showed hyperemia but no ulceration.

CASE 2.—C. J., a white infant, seven months old, was admitted on July 19, 1934, because of diarrhea for six days which had suddenly ceased the preceding day. Except for the fact that the infant had been treated for syphilis since birth, the family and past history were unessential. He suddenly became ill on July 13, with frequent, loose, greenish yellow stools. The stools contained no blood and averaged from six to eight per day. He took his feedings well and did not vomit until three days before admission when he seemed worse, became listless, and vomited most of his food. On July 18, two days later, he was brought to the out-patient clinic. The temperature was 37.8° C. (100° F.), he was moderately dehydrated, and a few fine râles were heard at both bases posteriorly though an x-ray picture of the chest showed no pneumonia. The mother was advised to force fluids and to return if the child did not improve. He had no stools for twenty-four hours, and when seen on July 19 he had a temperature of 41.1° C. (105.98° F.), was stuporous, dehydrated, and in a critical condition. No mass could be palpated in the abdomen. On rectal examination a rounded indefinite mass could be felt by the tip of the finger in the midline. On admission, at 5:00 P.M., the patient was given 150 c.c. 10 per cent glucose intravenously, and a continuous subcutaneous infusion of saline was started. The temperature dropped to 37° C. (98.6° F.). At 10:00 P.M. he vomited dark material with a nonfecal odor. A stomach tube was passed and left in situ. Enemas were ineffectual. An exploratory laparotomy was performed twelve hours after admission, and an ileocecal intussusception was found. This was easily reduced; the gut did not appear gangrenous. After the operation the child was given repeated infusions of glucose intravenously with some improvement. On July 22 he passed a stool from which *B. paratyphosus* B was cultured. He improved slightly until July 24, when the vomiting, nonfecal in character, recurred, and fine râles were heard at the bases of both lungs. A transfusion was given, but he died that evening. Postmortem examination showed fibrinous peritonitis and bronchopneumonia, but no recurrence of the intussusception.

CASE 3.—C. G., a five-month-old white infant, was admitted on July 26, 1934, because of vomiting, abdominal pain, and cessation of bowel movements. The family and past history were noncontributory. The patient was perfectly well until the day of admission. Then at 10:00 A.M. he suddenly doubled up and cried incessantly, apparently because of abdominal pain. At 3:30 P.M. he vomited after nursing; he continued to vomit yellowish green and later brown material. At 7:00 A.M. the following morning the patient had a normal bowel movement but later had two small movements consisting almost entirely of fresh blood. He was given several enemas, which were ineffectual. On admission the temperature was 38.8° C. (102° F.); he was acutely ill, in severe pain, but not dehydrated. His abdomen was slightly distended, but no loops of intestine could be seen. A cordlike structure

was palpated in the right lower quadrant along the border of the rectus muscle, but this was indefinite. There was tenderness throughout the abdomen on palpation, most marked in the right lower quadrant. Many fine gurgles could be heard, especially over the right lower quadrant of the abdomen. No masses could be felt by rectum, but bright red blood appeared as the examining finger was withdrawn. The child was immediately taken to the operating room where an intussusception at the ileocecal junction was easily reduced. He was given parenteral fluids following the operation. The temperature remained elevated, and for ten days he had from ten to eighteen stools daily, which contained much mucus but no blood. Parenteral fluids and blood transfusions were given as needed. *B. paratyphosus* B was isolated from the stools on the fourth day. The diarrhea gradually subsided, and the patient was discharged in good condition from the hospital twenty-four days after onset.

REFERENCES

1. Hess, J. H.: Arch. Pediat. **22**: 655, 1905.
2. Lichtenstein: Allg. med. Centr.-Ztg., Berl. **63**: 1894.
3. Fitz, R. H.: Acute Intestinal Obstruction, Boston, 1889, Cupples and Hurd.
4. Perrin, W. S., and Lindsay, E. C.: Brit. J. Surg. **9**: 46, 1921-22.
5. Moreton, A. L.: Brit. J. Surg. **7**: 409, 1919-20.
6. Madelung, O. W.: Chirurgie des Abdominaltyphus, Stuttgart, 1923, F. Enke.
7. Burhaneddin, A.: Zentralbl. f. Chir. **54**: 973, 1927.
8. Holzmann, E.: Deutsche med. Wchnschr. **59**: 1392, 1933.

AN UNUSUAL CASE OF GLOMERULAR NEPHRITIS WITH UREMIA

ROGER H. DENNETT, M.D., AND
CHESTER H. MYRON, M.D.
NEW YORK, N. Y.

EXTREME grades of uremia are uncommon in the glomerulonephritis of childhood although moderate nitrogen retention and hypertension are frequently noted. The following case is of interest because it presents one of maximal renal insufficiency with minimal clinical signs. Despite frequent periodic examination in the outpatient department, the gravity of his condition was not recognized until an acute exacerbation made admission to the hospital necessary. Following this were revealed for the first time the unusual features of this case which are presented below.

A white boy, nine and a half years old, was admitted to the wards July 17, 1934.

Past history included whooping cough at six months, chickenpox at seven months, mumps at four years, measles at six years, and a history of repeated low-grade upper respiratory infection covering the past five and a half years. He had never been a strong boy, was apathetic, played little with other children, and was regarded by his family as a peculiar child. His weight at three and a half years was 25 pounds; at five and a half years, 36.5 pounds; and on admission at nine years, 50 pounds; his height was 47.5 inches. For the past few years he had suffered so much from frontal headaches, constipation, anorexia, and vomiting and he was so thin and pale that it was difficult to determine just when the present illness began. His mother had brought him to the clinic at regular intervals, and there was little, if any, improvement in his condition.

Present Illness.—Seven days prior to admission his headaches became much more severe and constant, and vomiting occurred every morning and several times during the day. While previously he had been listless and apathetic, he was now drowsy and preferred to sleep a good deal during the day as well as at night.

Physical examination revealed a poorly nourished, underdeveloped boy, nine and a half years old; his pale face had a waxy tinge. He had a dull expression and seemed drowsy at times while being admitted. Respirations were deep but regular and of the normal rate. There was no edema of the face, extremities, or other parts. The nose, ears, mouth, and throat were negative, tonsils having been cleanly enucleated. The lungs were negative, and the heart was slightly enlarged to the left with a systolic murmur at the apex. The sounds were of good tone and the rate and rhythm were normal. The blood pressure was 128/82. The abdomen was negative; kidneys not palpated. Knee jerks and ankle jerks were hyperactive; there was a bilateral clonus and a normal plantar reflex.

From the Pediatric Department of the New York Post-Graduate Medical School of Columbia University.

Presented before the Regional Conference of the American Academy of Pediatrics in New York, Oct. 19, 1934.

Blood Chemistry.—On admission the urea nitrogen was 140.5; the nonprotein nitrogen, 183.5; the ratio between them, 76. The carbon dioxide combining power of the blood was 17.4. The albumin, globulin, and serum were normal throughout. Creatinine was high, 5.4, while the cholesterol was normal. The variation of these features of the blood chemistry are shown in Chart 1. The maximum urea nitrogen was 187.5. The maximum nonprotein nitrogen was 204.0, with a maximum ratio of 0.92. CO_2 for almost a month was between ten and eleven.

Urine.—He had two-plus protein constantly but seldom was there anything seen in the sediment. On one or two occasions there were from ten to twenty white blood cells and from fifteen to twenty red blood cells. His renal function test

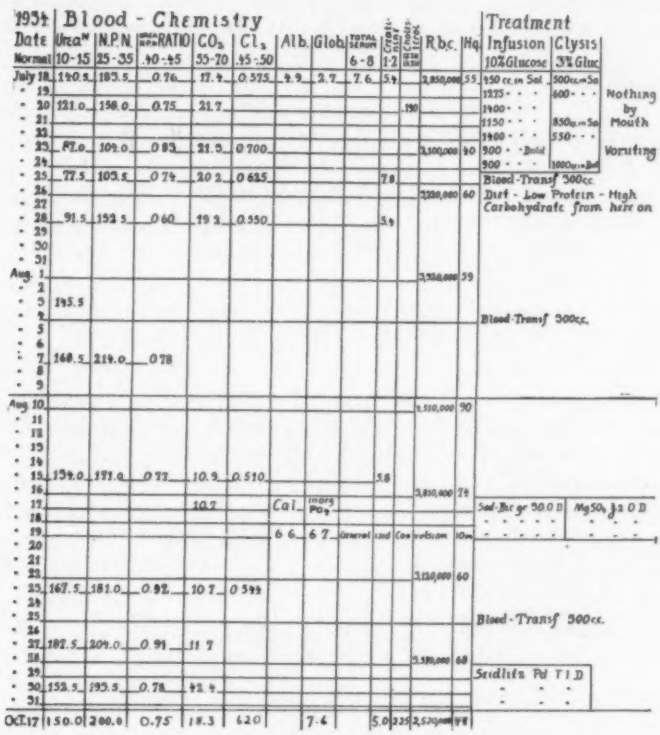


Chart 1.

(Chart 2) showed specific gravity varying from 102 to 110 as the maximum. The fact that the specific gravity never reached the normal point demonstrated markedly lowered renal function. In the twenty-four hours of the renal function test 1,040 c.c. of urine were voided.

Blood pressure was always somewhat high but never corresponding to the blood chemistry picture. The lowest found at any time was 106/72 and the highest, 128/82.

Progress Notes.—Following parenteral administration of glucose in saline solution, there was an increased output of urine, averaging 1,000 c.c. per twenty-four hours. Coincident with this there was a definite drop in urea and nonprotein nitrogen, with the ratio remaining constant. Clinically the boy was brighter and toleration of food by mouth returned.

With discontinuation of intravenous and subcutaneous therapy, the daily urinary output gradually became less and the urea, nonprotein, and ratio progressively rose. The carbon dioxide combining power became lower. One convulsion which occurred four weeks after admission was clonic in type and lasted for seven minutes. Five weeks after admission the urea nitrogen had risen to 204; the nonprotein nitrogen, 187.5; ratio, 92; carbon dioxide combining power, 11.7.

In an attempt to raise the carbon dioxide combining power of the blood ten grains of sodium bicarbonate three times a day were given for three days. The urine became strongly alkaline, but there was no corresponding rise in the carbon dioxide combining power. It was then deemed advisable to institute vigorous catharsis and diaphoresis on the basis that blood and tissue fluids were in equilibrium and that depletion of one would directly effect the other. Magnesium sul-

General Laboratory Data		Renal Function (2 hours)		
Age - 9 years		Time	Amount	Specific Gr.
Height - 47.5		7 AM - 9 AM.	100	1004
Weight - 47 to 50 lb		9 AM - 11 AM.	150	1006
Blood Pressure		11 AM - 1 PM.	100	1008
106 126		1 PM - 3 PM.	60	1002
72 80		3 PM - 5 PM.	50	1004
Urine		5 PM - 7 PM.	100	1006
Average Intake	1000cc - 1500cc.	7 PM - 7 AM.	480	1010
" Output	500cc - 700cc.			
" Specific Gravity	1002 - 1012			
" Protein	+++			
" Microscopic	Negative except 10-20 WBC & 15-20 RBC			

Chart 2.

FIRST TREATMENT		UREA*	N.P.N.	UREA/N.P.N. RATIO	INTAKE	URINE	DIAPHORESIS
DATE 8/30/34 TEMP. 100 to 105 FOR 3 HOURS	BEFORE	152.5	193.5	0.79	870cc.	200cc.	MODERATE
	AFTER	127.0	188.5	0.67			
SECOND TREATMENT							
DATE 9/5/34 TEMP. 100 to 103 FOR 3 HOURS	BEFORE	73.5	151.5	0.49	1195cc.	240cc.	MODERATE
	AFTER	111.5	154.5	0.71			
THIRD TREATMENT							
DATE 9/10/34 TEMP. 100 to 103 FOR 3 HOURS	BEFORE	103.5	146.5	0.70	1020cc.	190cc.	MODERATE
	AFTER	105.0	156.0	0.67			

Chart 3.

phate was first tried; since the patient vomited, one Seidlitz powder three times a day was substituted. At the end of one week the carbon dioxide combining power had risen to 51.3 from 11.7, and the urea had dropped to 73.5 from 204.0. Stools numbered one to two daily and were loose in character. The Seidlitz powder evidently had a twofold action, lowering the urea through elimination by bowel and raising the carbon dioxide combining power by increasing the available HCO_3 in the blood.

During this same period he was put in fever cabinet for four hours and body temperature by rectum maintained between 100 and 103° F. for three hours (Chart 3). There was a fall of 25 gm. in blood urea, and urea-nonprotein-nitrogen ratio fell from 0.75 to 0.67. Encouraged by this result, we repeated the treatment six days later, this time keeping him in the cabinet four and a half hours with a body temperature between 100 and 104° F. With the more vigorous hyperpyrexia

the urea rose from 73.5 to 111.5, and the ratio, from 0.49 to 0.71. Whether this paradoxical result was due to breaking down of body tissue at the higher temperature or to vasoconstriction of kidney blood vessels we cannot say.

Treatment.—For the first week the treatment consisted of 1,000 c.c. of 10 per cent glucose in saline injected intravenously and 500 c.c. of 3 per cent glucose in saline subcutaneously daily. Vomiting then ceased and the child maintained thereafter on a high CHO, low protein diet, and fluids forced by mouth. Three blood transfusions of 300 c.c. each were given at weekly intervals for the secondary anemia. Clinically the transfusions were apparently of more benefit than any other treatment since he became brighter, ate better, and looked better after each transfusion. The red blood cells gradually increased from 2,100,000 to 4,510,100, and the hemoglobin, from 40 per cent to 90 per cent.

SUMMARY

This case presents one of maximum renal insufficiency as shown by a persistent high urea nitrogen nonprotein nitrogen ratio. This ratio which normally averages 40 per cent has on several occasions been as high as 92 per cent. The highest blood pressure reading obtained was 126/82, and this is a definite, though moderate, elevation for a child nine years old. There was present a fairly severe secondary anemia, which responded best to the second blood transfusion. At no time was there any evidence of edema, a fact which can probably be accounted for by the fact that there has at no time been any depletion in the plasma proteins. That he is in a uremic state is shown by the tremendous nitrogenous retention in the blood, a persistent low carbon dioxide combining power of the blood, retention of inorganic phosphates, and a very high creatinine content. All of this evidence pointed to a chronic diffuse glomerulonephritis. The exact etiology in this case is uncertain. However, we can presume, from the history of repeated low-grade upper respiratory infections and periodic malaise covering almost five and one-half years, that the prime cause of his nephritis lay in these early episodes. He now looks fairly well, except for his pallor and is at home leading a moderately normal life in spite of the evidences of the blood chemistry, which is that of a uremic individual.

SPONTANEOUS HYPOGLYCEMIA DUE TO HYPERINSULINISM IN A CHILD

Report of a Case With Autopsy Findings

Arthur M. Dannenberg, M.D., M. A. Bell, M.D., and
Benjamin Gouley, M.D.
Philadelphia, Pa.

HYPOGLYCEMIA has been long recognized as occurring in functional disturbances of the liver and endocrine glands—notably the thyroid, pituitary, and adrenals—but it was not until 1924 when Seale Harris[1] attributed the chain of symptoms associated with hypoglycemia to hyperinsulinism that an active interest in this condition was aroused. Since then numerous articles on hyperinsulinism due to neoplasm or hyperplasia of the islet cells of the pancreas have appeared in the literature.

It was also in 1924 that Ross and Josephs[2] in a metabolic study of recurrent vomiting in children in which drowsiness and occasional convulsions occurred as part of the attack expressed their belief that hypoglycemia was probably an essential factor in these attacks. They quote Hilliger, who in 1914 had reached the same conclusion. Josephs[3] in 1929 also reported on ten cases of spontaneous hypoglycemia in childhood. In two cases that came to autopsy the liver was the site of extensive fatty change, a condition that was also present in all the reported cases that he was able to find in the literature. Griffith[4] in 1929 also attempted to show a relationship between convulsions in early life and hypoglycemia. In 1929 von Gierke[5] described a new clinical syndrome in children designated as "Hepatonephromegalia glykogenica," the characteristic findings of which are chronic hypoglycemia and hepatic enlargement due to excessive accumulation and fixation of glycogen in the liver and kidneys. Recently Kramer and others[6] reported on another type of hepatic hypoglycemia in a child; in this type they believed that the glycogen is not produced and is apparently replaced by fatty infiltration. The report of a case of spontaneous hypoglycemia by Rabinovitch and Barden[7] is of unusual interest in that the autopsy findings showed the medulla of the adrenal glands replaced by lymphoid tissue to such an extent that in all probability no adrenalin was secreted by the glands. These changes were believed to account for the hypoglycemia attacks in this patient.

From the Pediatric Department and Laboratories of the Jewish Hospital, Philadelphia.
Read before the Philadelphia Pediatric Society, Oct. 9, 1934.

In brief, hypoglycemia has been mainly attributed to severe structural and functional changes in the liver, to neoplasm, to hyperplasia of the islet cells of the pancreas, and to inadequate secretion of the medulla of the adrenal glands.

The symptoms of spontaneous hypoglycemia are characteristic. They include nervous irritability, anxiety, fatigue, hunger, tremor, muscular twitchings, lack of clearness of vision, diplopia, unsteadiness of gait, syncope, excess perspiration, loss of emotional control, convulsions, and coma. These symptoms may be due to so many other factors that the causal relation of spontaneous hypoglycemia may not be considered, unless blood sugar determinations are made.

The dramatic suddenness with which an attack of hypoglycemia may occur in apparently well children is illustrated in the four case reports of Elias and Turner[8] as well as in the following case report.

E. B., a thirteen-month-old white male child, was admitted to the Jewish Hospital on Dec. 1, 1931.

Past Medical History.—He was an only child born normally one month prematurely weighing 4 pounds. He was artificially fed from birth. He gained in weight slowly and always had a small capacity for food. At three months of age he had acute purulent otitis media; at six months, measles, and at eight months, chickenpox. He had never had a convulsion up to the time of his first admission. The first tooth appeared at ten months. He was unable to stand at thirteen months, when his weight was 16 pounds.

Family History.—Both parents were in good health. There was no history of diabetes on either side of the family.

History of Present Illness.—The evening before admission, the child took his supper of zwieback and milk. At 10:30 P.M. he drank 4 ounces of milk. He slept until 7 A.M., when he took but ½ ounce of milk. One-half hour later he vomited. Because of the large amount of vomitus, it was thought that some of the milk ingested the night before also came up. He became stuporous; he had an ashen color, cold and clammy skin, and a diverging strabismus.

Examination on admission to the hospital showed an underdeveloped stuporous child with an unusually small, slender, bony framework. The face was pale; the lips and nails cyanosed, and the skin, cold and clammy. During the examination he suddenly became rigid, with head thrown backward and the eyes rolled upward in a divergent squint. This attack lasted about thirty seconds. He made ineffectual attempts to vomit. The anterior fontanel was wide open. The conjunctivae were pale; the pupils were small, equal and reacted to light. Slight, vertical, nystagnoid movements were present. The ears, mouth, and pharynx were normal. A slight bilateral cervical adenopathy was present. The chest showed a slight rachitic rosary and flaring costal angle.

No pathologic process was determined present in the heart or lungs. The abdomen was scaphoid. The liver, spleen, and kidney were not palpable. Peristalsis was normal. Kernig, Chvostek, and Trousseau signs and nuchal rigidity were absent. The urine was entirely normal. Blood count: hemoglobin, 58 per cent; R.B.C., 4,350,000; W.B.C., 11,800; neutrophiles, 59 per cent; lymphocytes, 39 per cent; mononuclear leucocytes, 1 per cent; and transitional leucocytes, 1 per cent.

The tentative diagnosis at this time was (1) gastrointestinal upset, (2) early acute infection, and (3) possible drug poisoning.

After a colonic irrigation, the application of external heat, and glucose water by mouth, the child promptly improved. Within four hours, he was less stuporous and appeared like a child recovering from the effects of a sedative drug. Improvement continued. He was discharged three days after admission with a diagnosis of gastrointestinal toxemia. The possibility of the child's having hypoglycemic shock was not considered.

The *second admission* to the hospital was on Oct. 7, 1932, eleven months after the first, with a history of persistent vomiting the day before. On admission the child was stuporous and pale. The eyes were sunken and glassy, and the anterior fontanel, which was still open, was depressed. The hands were clenched, the arms held in abduction and flexed at the elbows. Carpopedal spasm was present. He had general muscular rigidity, catalepsy, and resisted passive movements. The pupils were small, equal, and reacted to light. Strabismus was present. The ears were normal. The tongue was coated and the breath foul. The pharynx was markedly congested. Except for a rapid rate, the heart was normal. The lungs were clear. The skin showed definite evidence of dehydration.

Impression: Infantile tetany from alkalosis due to persistent vomiting, gastrointestinal upset.

Laboratory Findings: Blood sugar content was too low to read; urea nitrogen, 17 mg. per cent; CO_2, 30 volumes per cent; creatinine, 1.3 mg. per cent; Urine acid S. G. 1.028; Albumin, very faint trace; sugar, negative; acetone, 4-plus; diacetic acid, negative.

From the laboratory findings, it was concluded that the cataleptic stuporous state was due to hypoglycemia. After the administration of 6 ounces of glucose water by mouth, the child rapidly came out of this stupor. Hypodermoclysis of 5 per cent glucose in normal saline solution brought about continued improvement. The day after admission the muscular rigidity, catalepsy, and strabismus were absent. He was discharged three days after admission with a diagnosis of acute gastrointestinal upset and hypoglycemia due to the persistent vomiting and lack of food.

The *third admission* was one month later, Nov. 7, 1932. In the interval the child was well; the morning of admission the mother noticed that on awakening, he seemed weak and unsteady when he attempted to stand. He vomited a small quantity of mucoid material. The mother gave him glucose water, but it was not retained. On admission to the hospital he presented a picture similar to that of his previous admission. His liver, however, was palpable three fingerbreadths below the costal margin.

Laboratory findings: Blood sugar, too low to read; calcium, 11 mg. per cent; phosphorus, 4.9 mg. per cent; urea nitrogen, 13 mg. per cent.

After absorbing 50 c.c. of 5 per cent glucose in normal saline solution, the child opened his eyes and began to cry. After 100 c.c. was absorbed, he was able to retain orange juice, after which the hypodermoclysis was discontinued.

The following day his condition was remarkably improved. His appetite was good. He seemed bright and cheerful.

A sugar tolerance test, November 9, using 1:75 gm. dextrose per kilogram of body weight, showed:

Fasting sugar level	71 mg. per cent
One-half hour	125 mg. per cent
One hour	120 mg. per cent
Two hours	120 mg. per cent

The urine was negative for sugar (twenty-four-hour specimen).

A surgical consultant advised against an exploratory operation for a possible pancreatic neoplasm.

Four days after admission the general condition was good. The blood Wassermann test was negative. The bromsulphalein test reported less than 5 per cent retention of dye at the end of one-half hour, which is normal. Several twenty-four hour specimens of urine were free from sugar. This definitely indicated that renal glycosuria was not the cause of the hypoglycemia.

On November 14, a week after admission, the patient's gait suddenly became uncertain like that of a baby learning to walk. A sudden squint also developed. The administration of orange juice brought about a prompt cessation of these symptoms.

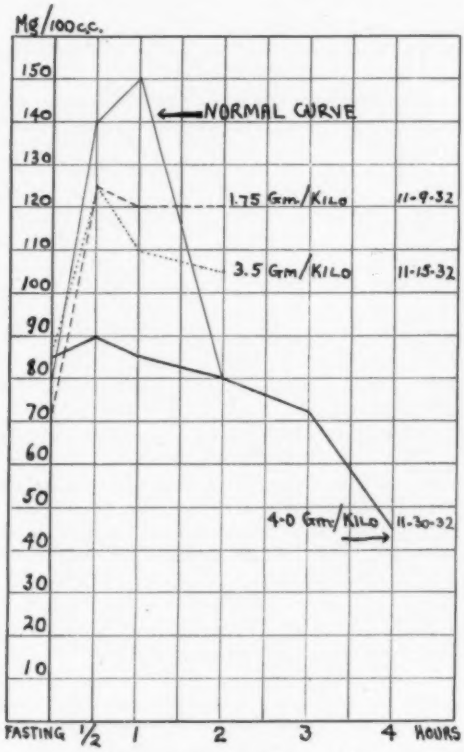


Chart 1.—Glucose tolerance tests.

A second glucose tolerance test, using 3.5 gm. per kilogram of body weight, showed:

Fasting sugar level	88 mg. per cent
One-half hour	125 mg. per cent
One hour	111 mg. per cent
Two hours	107 mg. per cent

The urine was negative for sugar (twenty-four-hour specimen).

Blood pressure was systolic 80, diastolic 48; roentgen ray examination showed a normal sella turcica. Tuberculin test was negative to 1 mg. of O.T.

On November 19 the patient had again a mild attack of hypoglycemic shock, which was relieved by orange juice. An attempt to determine the basal metabolism rate failed.

A third glucose tolerance test, November 30, using 4 gm. glucose per kilogram of body weight (more than twice the standard test dose) showed:

Fasting sugar	86 mg. per cent
One-half hour	91 mg. per cent
One hour	86 mg. per cent
Two hours	81 mg. per cent
Three hours	73 mg. per cent
Four hours	46 mg. per cent

The peak of the curve was 91 mg. per cent at one-half hour. At the fourth hour with a level of 46 mg. there were no clinical signs of hypoglycemia although the child was irritable and complained of hunger.

On December 4 the child was discharged with no definite improvement in his condition. The mother was opposed to an exploratory laparotomy for a suspected tumor or cyst of the pancreas.

Diagnosis: Hypoglycemic reaction due to hyperinsulinism.

The *fourth admission* on Dec. 12, 1932, was eight days following his discharge from the hospital. He had had frequent loose bowel movements for three days and vomiting for two days previous to admission. On admission he presented the same picture as before. The intravenous injection of a small amount of glucose solution promptly caused the child to sit up perfectly conscious. He was discharged two days after admission.

The *fifth admission*, Sept. 11, 1933, was nine months later. The child was carried into the accident ward, dead. During the nine months since his previous discharge, he had had but one attack of hypoglycemic shock, which occurred two weeks after his discharge from the hospital. For this he was treated at another institution. In February, 1933, he had bronchopneumonia with a temperature of 104° F. for over a week without developing any signs of hypoglycemia. He had been in fairly good health from that time until the morning of his death, when at 7 A.M. he suddenly had a convulsion. He was given glucose and orange juice by mouth but failed to regain consciousness. At 2:30 P.M. another convulsion occurred. He was rushed to the Jewish Hospital but died en route.

NECROPSY

Necropsy was performed fifteen hours after death; there was considerable rigor. The superficial lymph nodes were scarcely palpable.

Internal Examination.—There appeared to be marked dehydration. The subcutaneous fatty tissue was deficient. The serous cavities did not contain any fluid. The heart was normal except for the presence of a few small subendocardial hemorrhages in the left ventricle. The lungs were normal.

The thymus was perhaps enlarged; it weighed 25 gm., but the gland was normal in shape and consistency, and there was no evidence of compression of the adjacent structures.

The thyroid appeared to be definitely subnormal in size; it was of normal shape but weighed less than 5 gm. Section showed what appeared to be normal thyroid tissue with considerable colloid.

The spleen was grossly normal.

The pancreas was of normal size and consistency. Gross sectioning did not reveal any gross tumor formation. Two small, rounded, grayish bodies were found on the superior surface of the tail of the organ; these were apparently small lymph nodes.

The adrenals were apparently diminished in size. Section showed a narrow but well-preserved cortex and a widened and well-preserved medulla.

The liver was of normal size and consistency. Section showed some areas of pallor, apparently parenchymatous degeneration in which lobulation seemed to be entirely lost.

The kidneys were grossly normal. The gastrointestinal tract was normal throughout.

The brain revealed no gross abnormality. The pituitary was of normal size and shape.

Histopathology.—Heart: The myocardium showed an acute granular degeneration in the majority of fields. There was no cellular infiltration; the change appeared to be purely parenchymatous.

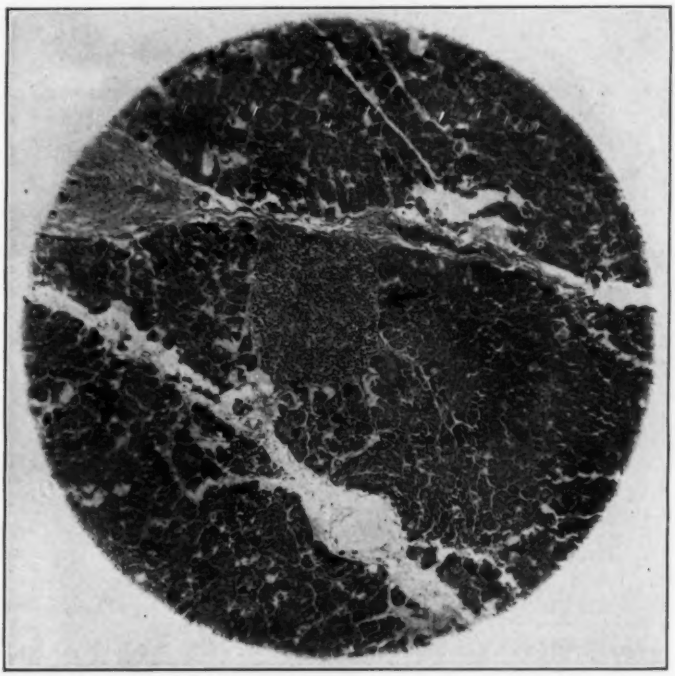


Fig. 1.—Showing large island of Langerhans consisting of densely packed islet cells. Note absence of the normal trabecular or cord formation.

Thymus: Well-preserved thymic tissue of normal type was present.

Thyroid: Two sections showed an inactive structure; numerous acini were filled with homogeneous dark stained colloid; the lining epithelium was flattened.

Adrenals: The cortex was normal, in some places it appeared to be narrow, in contrast to which the medulla was distinctly hyperplastic.

Liver: The architecture was normal. There was acute congestion. The individual cells showed a marked granularity of their cytoplasm. Their boundaries were well outlined, in some places sharply so. There was no vacuolation. In some fields there was early nuclear (necrobiotic) changes such as pyknosis.

Pituitary: The pituitary was entirely normal.

Pancreas: Sections taken from numerous blocks throughout the pancreas showed the following changes: (1) hyperplasia of the islands of Langerhans (Fig. 1); (2) a periductile fibrosis more noticeable in the head and body of the organ (Fig. 2); and (3) an acute necrosis in the head of the pancreas associated with dilatation and catarrhal obstruction of the pancreatic ducts.

1. The changes in the islands of Langerhans were seen best in the tail, but hyperplastic islands were occasionally seen in most of the sections of the entire organ. The hyperplasia was not in the number of islands, which apparently was normal, but in the changes in the individual islands. They were of two types: (a) the more normal type in which the island cells had maintained to some degree

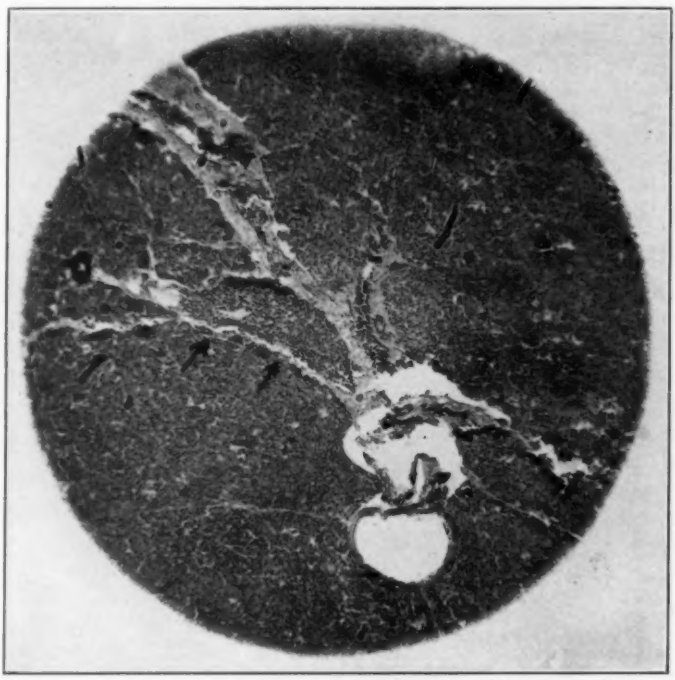


Fig. 2.—Large atypical island of closely clustered islet cells. Note also the increased periductile fibrosis.

their cordlike or trabecular arrangement of cells with proportional capillary representation and (b) much more numerous islands showing definite cellular hyperplasia. The cellularity even in the slightly enlarged islands was greatly increased so that the original trabecular arrangement was lost, the cells being tightly packed against each other. In some islands the cells appeared to be in large solid clusters. The size of the islands varied; even in the head of the pancreas some abnormally large islands were noted. The largest were seen in the tail where they may be three or four times the size of normal. The nuclei of these very cellular islands stained deeply; many of them could be described as hyperchromatic. None, however, showed definite evidence of rapid cell division. The islands, therefore, appeared to be hyperplastic but not definitely neoplastic. In some islands where

the cellular concentration was not so great, the cytoplasm could be seen to better advantage; it appeared to be markedly granular.

2. Periductile fibrosis was present and accompanied by a mild interlobular fibrosis. This change, best seen in the proximal half of the organ, is unusual for a patient of this age.

3. In the head of the organ there was an acute necrotic degeneration, spreading backward and irregularly through the body. The necrosis was associated with, and apparently secondary to, an acute dilatation and obstruction of the pancreatic duct at and near the ampulla. There was a mild but definite catarrhal duodenitis. The pancreatic duct was filled with cellular débris; its mucosa had been destroyed, and destructive catarrhal occlusion of the duct could be followed in the branches throughout the body. Necrosis was seen in the head, but seminecrotic changes and edematous swelling of the lobules could be seen throughout the body; the blood vessels appeared to play no part in the process.

Although the patient was dead fifteen hours before necropsy, sections of liver were stained for glycogen by the Best carmine stain; one small drop of the glycogen was noted after reviewing many sections. As a control experiment, the liver of an adult diabetic patient whose necropsy was also performed sixteen hours after death was examined and was found to be loaded with glycogen. While the rather late hour of necropsy precludes any definite statement as to glycogen content in this case, this contrast with a known case of glycogen storage was considered to be of interest.

DISCUSSION

This case may be regarded as one of spontaneous hypoglycemia due to hyperinsulinism. It serves to call attention to the fact that in children sudden weakness, muscle tremor, strabismus, convulsions, and coma may be due to hypoglycemia and that blood sugar determinations should be routinely performed when such symptoms arise.

The stimulating effect of glucose ingestion on the child's insulogenic apparatus is evident from a study of his glucose tolerance tests. With increasing amounts of glucose, a lower blood sugar curve occurred. When 4 gm. of glucose per kilogram was ingested, a fasting blood sugar of 86 mg. per cent rose in one-half hour to only 91 mg. per cent and then gradually diminished to 46 mg. per cent at the end of four hours when he showed the unexplained paradox of having no symptoms of hypoglycemia.

The depressing effect of an infection upon insulin production was strikingly evident from the fact that no attacks of hypoglycemic shock occurred during an attack of severe bronchopneumonia, when because of the increased metabolism and lessened food intake such attacks would be expected to take place.

The cause of the cellular hyperplasia of the individual islands of Langerhans of this case is not known. The parents are not diabetic. Gray and Feemster[9] reported an interesting case of hypertrophy and hyperplasia of the islands of Langerhans in the pancreas of a child born of a diabetic mother whose diabetes became much milder during the last month of pregnancy and more severe after parturition. The

infant on the third day of life had a blood sugar content of 67 mg. per cent and died on the fourth day. The islet tissues of this infant's pancreas were twenty-four times as great as normal.

It is possible that our patient may have had diabetes very early in life; unfortunately we have no way of proving or disproving this possibility. Boyd and Robinson[10] reported what they consider an instance of regeneration taking place in the islands of the pancreas of a child dying by accident, who had been treated with insulin over a period of seven years with gradual improvement of diabetes. There were numerous small bulbous islands resembling the regenerative islands seen in experimental animals, and, since the necropsy was held immediately, it was definitely determined that the new islands were composed almost exclusively of beta cells. The comparatively late hour of necropsy in our case did not permit such accurate cellular identification.

In this case the possibility of dysfunction of the other endocrine glands as a factor in the production of hypoglycemia is necessarily very speculative. The appearance of the pituitary gland was entirely normal both as to its size and also as to the ratio of the eosinophilic to the basophilic cells. The thyroid gland was small even for the age of this patient. The histology was that of an inactive gland, but it would be difficult to state definitely that an element of hypothyroidism was present. In this connection it is well to remember, however, that low blood sugar determinations are not uncommon in hypothyroidism and myxedema. Our patient did not have the clinical picture of myxedema. The adrenal glands showed a well-preserved and apparently hyperplastic medulla. The antagonism of adrenalin and insulin is well known. It is possible that in our patient there was some degree of physiologic compensation on the part of the adrenal medulla in an effort to ward off hypoglycemia. If this were so, one might assume that the adrenal medullary hyperplasia eventually failed in its function. There was nothing in the histology of the liver to suggest a hepatic factor in the establishment of the hypoglycemia. The bromsulphalein test was normal. It is to be regretted that necropsy was performed fifteen hours after death. The entire absence of glycogen in the liver, therefore, may or may not be of significance.

Treatment of the hypoglycemic attacks is relatively simple and consists of the oral or parenteral administrations of sugar. The prevention of subsequent seizures depends upon a determination of the cause. This is extremely difficult; surgical intervention must be considered and is, according to Judd and his coworkers,[11] eminently successful if a localized islet tumor can be removed from the pancreas. In some cases[12] in which a normal pancreas was found at operation and was partly resected, a partial and temporary relief from symptoms was obtained.

A low-carbohydrate, high-fat diet has been recommended in order to avoid the stimulating effect of carbohydrate on insulin production. Recently H. J. John[13] has recommended the use of insulin in the treatment of hyperinsulinism. He has obtained excellent results with this treatment, which he explains as follows:

"In the morning patients have a low blood sugar. After meals the blood sugar rises, and, when it reaches a certain height, this postprandial hyperglycemia stimulates the pancreas to pour out insulin into the blood stream, and the blood sugar begins to drop. Normally the blood sugar reaches a certain level below 120 mg. per cent at which time the insulin supply is automatically decreased or discontinued. In patients with hyperinsulinism, however, this regulation does not take place, and the blood sugar continues to fall to a very low level when symptoms of hypoglycemic shock or insulin reaction appear. By giving 10 units of insulin to such a patient three times a day, one-half hour after each meal, the following may be accomplished: One-half hour after meals the blood sugar has not yet reached the upper level of stimulation of the islands of Langerhans and injected insulin should prevent its reaching this level. With the exogenous insulin in the blood stream, hyperglycemia fails to develop, and there is no stimulation for the excessive output of endogenous insulin which in the untreated patient causes the lower level of shock to be reached. If the dosage is properly adjusted and the patient does not get insulin from the pancreas, then the drop of his blood sugar should not reach the lower level at which the insulin reaction becomes manifest. In this manner the patient's blood sugar level is kept between the upper and lower levels. The pancreas is placed at physiologic rest and hypoglycemia is eliminated."

SUMMARY

A physically underdeveloped male child had been subject since the age of thirteen months to attacks in which he would suddenly become weak, pale, and then comatose with strabismus, general muscular rigidity, catalepsy, and an occasional convulsion. He would rapidly recover from these attacks upon the administration of sugar orally or parenterally. Laboratory studies revealed the attacks to be due to a low blood sugar level. The sugar tolerance was increased, and there seemed to be a failure of normal balance between the factors that control the blood sugar level. The evidence pointed to a state of hyperinsulinism, the cause of which was not determined during the patient's life. He died at the age of three years from what appeared to be hypoglycemic shock. Autopsy findings determined a cellular hyperplasia of the individual islands of Langerhans.

REFERENCES

1. Harris, Seale: J. A. M. A. **83**: 729, 1924.
2. Ross, S. G., and Josephs, H. W.: Am. J. Dis. Child. **28**: 447, 1924.
3. Josephs, H. W.: Am. J. Dis. Child. **38**: 746, 1929.
4. Griffith, J. P. C.: J. A. M. A. **93**: 1526, 1929.
5. Von Gierke, E.: Beitr. z. Path. Anat. u. z. allg. Path. **82**: 497, 1929; and Med. Klin. **27**: 576, 611, 1931.
6. Kramer, B.; Grayzel, H. G.; and Solomon, C. I.: J. PEDIAT. **5**: 299, 1934.
7. Rabinovitch, J., and Barden, F. W.: Am. J. M. Sc. **184**: 494, 1932.
8. Elias, H. L., and Turner, R.: J. A. M. A. **98**: 2198, 1932.
9. Gray, S. H., and Feemster, L. C.: Arch. Path. & Lab. Med. **1**: 348, 1926.
10. Boyd, G. L., and Robinson, W. L.: Am. J. Path. **1**: 135, 1925.
11. Judd, E. S., Allan, F. M., and Rynearson, E. H.: J. A. M. A. **99**: 102, 1933.
12. Finney, J. M. T., and Finney, J. M. T., Jr.: Ann. Surg. **88**: 584, 1928.
13. John, H. J.: M. Clinic North America **17**: 979, 1934.

235 SOUTH FIFTEENTH STREET

INFANT MORTALITY IN NEW YORK CITY ONE HUNDRED YEARS AGO

CHARLES BOLDUAN, M.D., AND LOUIS WEINER
NEW YORK, N. Y.

FROM the many papers which have been published regarding the reduction of infant mortality during the past twenty or thirty years, one gains the impression that infant mortality rates a hundred years or more ago must have been frightfully high and that a fairly continuous reduction has taken place since that time. We believe this impression to be erroneous.

On the basis of a careful analysis of available statistics and despite the incompleteness of certain basic data, we have attempted to trace the approximate course of the infant mortality rate in New York City throughout the nineteenth century. The picture may come as a surprise to many who have been active in the intensive campaign for the reduction of infant mortality during the past twenty or thirty years.

SOURCE OF THE DATA

The deaths recorded in the official city publications, the so-called "City Inspectors' Reports" are admittedly incomplete. They were compiled regularly from the interment records of the various cemeteries. It is probable, however, that for the first quarter of the nineteenth century the number of deaths thus registered is not much below the actual number which occurred. A number of the "City Inspectors' Reports" during the *second* quarter of the century, do, to be sure, lament the fact that deaths in which interment occurred outside of the city fail to be registered among the city deaths. Nevertheless, such out-of-town interments were probably relatively few during the first quarter of the century, for there were sufficient cemeteries in and near the built-up portions of the city, and, on the other hand, ferry facilities whereby Long Island or New Jersey could be reached for interment were primitive and meager.

NUMBER OF BIRTHS

In order to calculate an infant mortality rate we now insist on a registration of the births. Such a record was not established in New York City until 1853, and even then for many decenniums later, this record was notoriously incomplete. Lacking a registration of births one can base an infant mortality rate on the population under one, as

From the Department of Health, City of New York.

determined by census enumerations. This method gives useful information, though it is not entirely accurate. Unfortunately, we are unable to use even this method for New York City for the first quarter of the nineteenth century owing to the manner in which the federal census enumerations were made prior to 1850. Thus the enumerations of 1810 and 1820 give the number of children of whites only "under ten," and for free colored and slaves only "under fourteen." In the enumerations of 1830 and 1840 there are figures for "under five" for whites, but only for "under ten" for free colored and slaves. The enumeration of 1850 is the first in which the population under one is given.

From the population under five years of age as shown by the census enumerations of 1830 and 1840 and with the aid of the recorded deaths under five years, we can make an estimate of the probable number of births during the preceding five years. On this basis it would appear that in the quinquennium 1826-1830 the birth rate in New York City was approximately 39 per 1,000, and in 1836-1840 it was about 43 per 1,000. Such birth rates agree very well with those prevailing at that time in European countries from which statistics are available. Thus, during the fourth decade of the nineteenth century the birth rate was 40 in Prussia, 40 in Wuerttemberg, and 32 in Sweden. During the sixth decade it was 34 in England and Wales. Moreover, birth rates of about 40 are only slightly higher than those which on the basis of other methods of calculation, we believe, prevailed in New York City even during the second half of the nineteenth century.

While it is impossible to get much information regarding birth rates in American communities during the early part of the nineteenth century, we do have data here and there which indicate that the birth rate was high. Thus in the *Medical Repository* for 1810 we find that in 1809 Portsmouth, N. H., with a population of approximately 7,000, had 289 births, a rate of over 41 per 1,000, and Newport, R. I., with about the same population, 260 births, a rate of over 37 per 1,000.

If we assume that the birth rate in New York City in 1806-1810 was 40 per 1,000, the *recorded* infant deaths during that period would yield an infant mortality rate of 113 per 1,000 births. Even adding 10 per cent to this to compensate for incomplete registration of deaths, the rate would be about 125 per thousand births.

COURSE OF INFANT MORTALITY

In Chart 1 we show approximate infant mortality rates in New York from 1804 to date. During the earlier years we have estimated our infant population on an assumed birth rate of 40 per thousand. In recent years, we have, of course, based the rate on the number of reported births. Where census enumerations made available the popu-

lation under five years, we have calculated the probable number of births and indicated the resulting infant mortality rate as a check on the other figures.

It will be noted that, contrary to popular conceptions, the infant mortality rate in New York City during the early part of the nineteenth century was relatively low. On the basis of a birth rate of 40

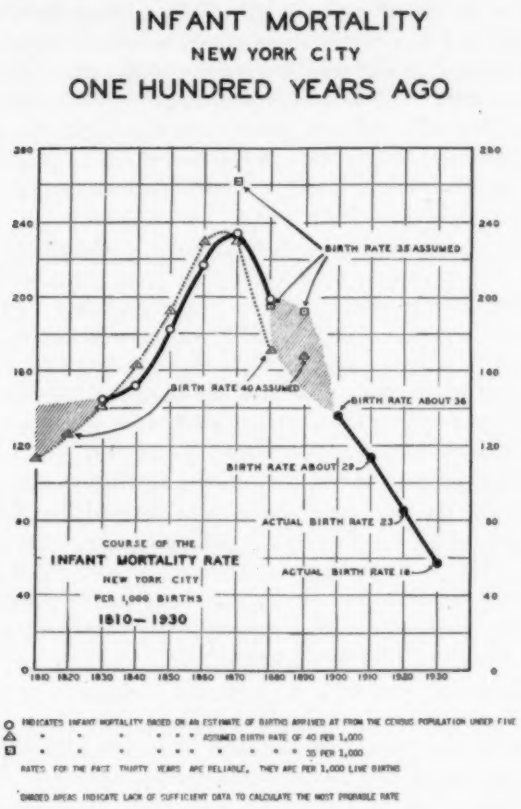

INFANT MORTALITY
NEW YORK CITY
ONE HUNDRED YEARS AGO
BIRTH RATE 35 ASSUMED
BIRTH RATE 40 ASSUMED
BIRTH RATE ABOUT 36
BIRTH RATE ABOUT 29
ACTUAL BIRTH RATE 23
ACTUAL BIRTH RATE 18
COURSE OF THE
INFANT MORTALITY RATE
NEW YORK CITY
PER 1,000 BIRTHS
1810—1930
280
240
200
160
120
80
40
0
1810 1820 1830 1840 1850 1860 1870 1880 1890 1900 1910 1920 1930
INDICATES INFANT MORTALITY BASED ON AN ESTIMATE OF BIRTHS ARRIVED AT FROM THE CENSUS POPULATION UNDER FIVE
ASSUMED BIRTH RATE OF 40 PER 1,000
35 PER 1,000
RATES FOR THE PAST THIRTY YEARS ARE RELIABLE, THEY ARE PER 1,000 LIVE BIRTHS
SHADED AREAS INDICATE LACK OF SUFFICIENT DATA TO CALCULATE THE MOST PROBABLE RATE

Chart 1.

the infant mortality rate calculated on the recorded deaths would be under 120, and, if we assume a birth rate of only 35, the rate would appear to have been not over 130 per 1,000 births. Even allowing that there was some incompleteness in the reported deaths, we find the infant mortality rate at the beginning of the nineteenth century probably did not exceed 145 per 1,000 births. Following the enactment of legislation in 1853 providing for the registration of deaths, the rates beginning 1860 are based on complete mortality returns.

The chief point of interest in this chart, and the main object of this paper, is to bring out the fact that, from a relatively low infant mortality rate at the beginning of the nineteenth century, the rate rose markedly to a maximum during the sixties and that it thereafter declined. The prevalence of low rates during the earlier years must, we feel, be accepted as a fact, although naturally the actual figures can be regarded as only approximately correct. A consideration of the local situation supports the validity of the picture thus brought out.

During the first quarter of the nineteenth century New York City was chiefly engaged in shipping and trade. Manufacturing played only a very minor rôle. The artisans were largely independent workers; steam-power factories did not become common until the forties and fifties. Housing conditions were favorable; there were no tenements. Overseas immigration did not get well under way until the 1830's, when it began to be a problem.

The second quarter of the century brought profound changes. The population growth became accelerated; factories multiplied; immigration from Europe increased rapidly, especially during the late forties. The competition of the ever increasing immigrants lowered all standards of living. Wages, especially after the panic of 1837, were reduced, and again reduced. The housing situation became acute. To relieve the overcrowding the cheapest kinds of houses were built. All in all, conditions became more and more insanitary.

The built-up portion of the city had meantime extended rapidly. The character of the city's milk supply had deteriorated, for there was little or no pasturage for the cows in the city dairies. Throughout the 1840's and '50's the insanitary condition of the city dairies and the almost general adulteration of the milk repeatedly engaged the attention of the authorities and of committees of public-spirited citizens. The infant mortality rate continued to rise.

During the 1850's and '60's the lack of an efficient system of health administration, repeatedly brought out, led finally to legislative action organizing the present Department of Health and granting wide powers to the Board of Health. Considerable attention was paid by the new Department of Health to sanitation, street cleaning, and housing. From all accounts the health work was efficient. After 1870 the infant mortality rate began to decline.

It is worthy of mention that in 1876 the Board of Health, impressed by the high infant mortality, obtained a special appropriation for the employment of a special staff of physicians during the months of July and August. This staff, known as the "Summer Corps" canvassed the congested tenement districts treating all sick babies whose parents were unable to obtain medical care. This plan of dealing with infant mortality was followed for many years.

The last two decades of the nineteenth century ushered in scientific methods for the reduction of infant mortality, methods based largely on the epoch-making discoveries of Pasteur and Koch. The infant mortality rate continued to decline so that despite the enormous congestion of population the admittedly insanitary conditions prevailing in large parts of the city, and the very great degree of industrialization, the rate at the close of the nineteenth century was again as low as it was a century before.

The course of the infant mortality rate since 1900 is so well known that it needs no discussion. The improvements in the milk supply, the establishment of baby health stations, the organized educational efforts, the compulsory pasteurization of milk, the great decrease in flies resulting from the introduction of motor cars—all these have been important factors in the further reduction of infant mortality, and their rôle has been well recognized.

SUMMARY

Despite the lack of important statistical data, a picture is presented showing the course of the infant mortality rate in New York City throughout the nineteenth century.

Evidence is presented to show that in New York City the infant mortality rate at the beginning of the nineteenth century was probably no higher than it was at the opening of the present century.

That the picture is approximately correct is evidenced by a discussion of the local conditions.

IS THE BENEFIT OF LACTIC ACID MILK IN INFANT FEEDING DUE TO THE BACTERICIDAL ACTION OF THE LACTIC ACID?

KENNETH B. ROTHEY
DURHAM, N. C.

THE popularity of buttermilk as an infant food is attested by the number of explanations offered for its success: sterility of milk, low fat, effect of lactic acid on metabolism, gastric and intestinal asepsis, effect on peristalsis, effect of the food combination on metabolism, and combinations of these.[1] Nil Rosen von Rosenstein[2] first recommended buttermilk as an infant food in 1776, but it soon fell into oblivion and was not again introduced into Holland until 1865,[3] in Germany until 1902,[4] in France until 1907,[5] and in the United States until 1919.[6]

Whole lactic acid milk, which at present probably is one of the best artificial infant foods, is of three varieties: (1) whole lactic acid evaporated milk; (2) whole lactic acid cultured milk; and (3) whole lactic chemical milk. All three are satisfactory, but the first is the cheapest; the second the easiest to use (if it can be purchased from a dairy); and the third a good substitute if either of the others is not available.

Whole lactic acid evaporated milk containing 7.5 per cent of added carbohydrate is made by diluting unsweetened evaporated whole milk with an equal amount of cooled, boiled water, to which previously have been added 1.5 per cent lactic acid (U. S. P.) and 15 per cent cane sugar. (A final dilution of 0.75 and 7.5 per cent, respectively.)[7] Whole lactic acid cultured milk is pasteurized grade A or B milk which has been inoculated with a culture of *B. bulgaricus* or *B. acidophilus*, and *Streptococcus acidi lactici* at the dairy, or in the home, and incubated at 37° C. (98° F.) overnight. Whole lactic acid chemical milk is made by adding, drop by drop, one teaspoonful of lactic acid (U. S. P.) or one tablespoonful of lemon juice to one pint of pasteurized grade A or B milk, while it is being stirred constantly. Additional carbohydrate (7.5 per cent) may be added to either of the two latter types of milk. The reason for adding 7.5 per cent carbohydrate to whole lactic acid milk is that infants usually are constipated if the amount is less and their stools generally are "loose," if more. Furthermore, the caloric calculations are greatly simplified by the addition of exactly 7.5 per cent sugar, for the resulting mixture then contains one calorie per cubic centimeter. The addition of carbohydrate to lactic acid milk is unnecessary for infants over the age of eight months.

From the Departments of Pediatrics and Bacteriology, Duke University School of Medicine.

To determine whether lactic acid milk is bactericidal, five series (A to E) of ten tubes each were set up. In each series, tubes 1 to 6, inclusive, contained 10 c.c. of one of six different varieties of lactic acid milk; tube 7 contained 10 c.c. of lemon juice milk; tube 8, 10 c.c. of egg white in water (1:25 dilution); tube 9, 10 c.c. of breast milk; and tube 10, 10 c.c. of broth. Each of these ten tubes was inoculated with 0.1 c.c. of an eighteen-hour broth culture of one of the following organisms: *B. dysenteriae* (Shiga) in series A; *B. dysenteriae* (Flexner) in series B; *B. typhosus* in series C; *B. paratyphosus A* in series D, and *B. paratyphosus B* in series E. All of the tubes were then incubated at 37° C. At intervals for the next fourteen hours a loopful of the contents of each tube of series A and B was subcultured on plates of Teague's medium, and of series C, D, and E, on brilliant green media. These plates were then incubated at 37° C. for eighteen hours, and the number of colonies were counted and recorded in Table I.

DISCUSSION

As may be seen in Table I, the addition of 1.1 per cent lactic acid to evaporated milk prevented the growth of *B. dysenteriae* (Shiga) and killed the other organisms in less than six hours. Somewhat similar results were obtained with cultured lactic acid milk and Mead's lactic acid milk No. 2. In the other mixtures (Mead's lactic acid milk No. 1, whole cow's milk containing 3 per cent lemon juice, egg medium, breast milk, and broth medium), there was luxuriant growth.

Obviously milk containing 1.1 per cent lactic acid is most desirable from a bactericidal point of view, but children do not like it because of its extremely sour taste. Milk containing 0.92 per cent and 0.66 per cent of lactic acid, however, is palatable and is sufficiently bactericidal to prevent chance infection, although six hours may be necessary to produce sterility. It is true that some children who previously have been fed with sweet milk mixtures at first may dislike the taste of the lactic acid milk, but no infant at Duke Hospital in the past two years has refused to take a mixture containing 0.75 per cent lactic acid provided that the nurse persisted in feeding it for forty-eight hours. In fact, several such children, after they had become accustomed to the whole lactic acid evaporated milk, refused to drink sweet milk mixtures. The feebleness of the bactericidal strength of lysozyme reported to be contained in egg white and breast milk[8] is demonstrated by the luxuriant growth in tubes 8 and 9.

CONCLUSIONS

1. Milk containing 0.66 per cent, or more, of lactic acid is bactericidal and will prevent its contamination with dysentery and typhoid organisms.

TABLE I

EFFECT OF LACTIC ACID ON PATHOGENIC INTESTINAL BACILLI*

LENGTH OF INCUBATION IN HOURS	TUBE 1	TUBE 2	TUBE 3	TUBE 4	TUBE 5	TUBE 6	TUBE 7	TUBE 8	TUBE 9	TUBE 10
	EVAPORATED MILK AND WATER (EQUAL PARTS)			CULTURED WHOLE LACTIC ACID MILK	MEAD'S POWDERED LACTIC ACID MILK NO. 2, LACTIC ACID, 0.5%	MEAD'S POWDERED LACTIC ACID MILK NO. 1, LACTIC ACID, 0.3%	WHOLE COW'S MILK, LEMON JUICE, 3%	EGG WHITE 1:25 DILUTION	BREAST MILK (CONTROL)	BROTH MEDIA (CONTROL)
	LACTIC ACID (U.S.P.), 1.1%	LACTIC ACID (U.S.P.), 0.9%	LACTIC ACID (U.S.P.), 0.66%							
Series A—inoculated with B. dysenteriae (Shiga)										
½	0	+	+	+	+	+	++	+	+	+
1	0	+	+	+	+	++	++	+	+	+
2	0	+	+	+	++	+++	++	+++	+++	+++
6	0	0	0	0	0	+	+++	++++	++++	++++
10	0	0	0	0	0	0	++++	++++	++++	++++
14	0	0	0	0	0	0	++++	++++	++++	++++
Series B—inoculated with B. dysenteriae (Flexner)										
½	+	+	+	+	+	+	+	+	+	+
1	+	+	+	+	+	+	++	+	+	+
2	+	+	++	++	+	+	+	++	++	++
6	0	0	0	+	0	+	++	++++	++++	++++
10	0	0	0	0	0	+	++++	++++	++++	++++
14	0	0	0	0	0	0	++++	++++	++++	++++
Series C—inoculated with B. typhosus										
½	+	+	+	+	+	+	++	+	+	+
1	+	+	+	+	+	++	++	++	+	+
2	+	++	++	++	++	++	+++	+++	+++	+++
6	0	+	+	+	++	+++	++++	++++	++++	++++
10	0	0	0	0	+	+++	++++	++++	++++	++++
14	0	0	0	0	0	++++	++++	++++	++++	++++

*0=sterile; +=1-5 colonies; ++=5-25 colonies; +++=25-50 colonies; ++++=plate covered with colonies.

TABLE I—CONT'D

LENGTH OF INCUBATION IN HOURS	TUBE 1	TUBE 2	TUBE 3	TUBE 4	TUBE 5	TUBE 6	TUBE 7	TUBE 8	TUBE 9	TUBE 10
	EVAPORATED MILK AND WATER (EQUAL PARTS)			CULTURED WHOLE	MEAD'S POWDERED	MEAD'S POWDERED	WHOLE COW'S MILK,	EGG WHITE	BREAST	BROTH
	LACTIC ACID (U.S.P.), 1.1%	LACTIC ACID (U.S.P.), 0.9%	LACTIC ACID (U.S.P.), 0.66%	LACTIC ACID MILK	LACTIC ACID MILK NO. 2, LACTIC ACID, 0.5%	LACTIC ACID MILK NO. 1, LACTIC ACID, 0.3%	LEMON JUICE, 3%	1:25 DILUTION	MILK (CONTROL)	MEDIA (CONTROL)
Series D—inoculated with B. paratyphosus A										
½	+	+	+	+	+	+	+	+	+++	++
1	+	+	+	+	+	+	+	++	++++	+++
2	+	++	++	++	++	++	+++	+++	++++	++++
6	0	++	++	++	++	+++	++++	++++	++++	++++
10	0	0	+	+	+	++++	++++	++++	++++	++++
12	0	0	0	0	0	++++	++++	++++	++++	++++
Series E—inoculated with B. paratyphosus B										
½	+	+	+	+	+	+	+	+	++	+
1	+	++	+	+	+	++	++	++	++	++
2	+	++	++	++	+++	+++	+++	+++	+++	+++
6	0	+	+	+	++	++++	++++	++++	++++	++++
10	0	0	0	0	0	++++	++++	++++	++++	++++
12	0	0	0	0	0	++++	++++	++++	++++	++++

2. Infants will drink milk containing 0.75 per cent lactic acid.

3. The use of bactericidal or antiseptic milk should be encouraged to prevent milk-borne infections.[9] This bactericidal action also is useful in reducing the growth of bacteria in the upper intestines which accompanies diarrhea.

4. The home instructions for preparing lactic acid evaporated milk are simple: The contents of a 7-cent can of unsweetened evaporated milk (390 c.c., 13 oz.) are poured into a quart bottle, previously scalded with boiling water, and the empty can nearly filled with boiling water, in which then are dissolved one teaspoonful of lactic acid (U. S. P.), and three level tablespoonfuls of cane sugar. As soon as this solution is cold, it is mixed slowly, being stirred constantly, with the unsweetened evaporated milk. In order for the lactic acid to accomplish its bactericidal effect, the mixture should be kept for six hours, preferably, though not necessarily, in a cold place before being fed in required amounts to the infant. After being mixed, it should not be repasteurized or boiled, as it will curdle.[7] This mixture (810 c.c., 27 oz.) is sufficient for a twenty-four-hour diet for any infant, regardless of age. Older children, of course, require solid food in addition.

REFERENCES

1. Gerstley, J. R.: Am. J. Dis. Child. 45: 538, 1933.
2. Von Rosenstein, N. R.: Diseases of Children and Their Remedies, London, 1776, Cadell, p. 62.
3. Ballot, A. M.: Nederl. tijdschrift v. geneesk. 2: 402, 1865.
4. De Mattos, T.: Jahrb. f. Kinderh. 55: 1, 1902.
5. Pehu, M.: Lyon méd. 109: 837, 1907.
6. Marriott, W. McK.: J. A. M. A. 73: 1173, 1919; Idem, and Schoenthal, L.: Arch. Ped. 46: 135, 1929.
7. Davison, W. C.: The Compleat Pediatrician, Durham, 1934, Duke University Press.
8. Rosenthal, L., and Lieberman, H.: J. Infect. Dis. 48: 226, 1931.
9. Davison, W. C.: Medicine 1: 389, 1922.

BACTERIOLOGIC STUDIES OF THREE SMALL EPIDEMICS OF INFECTIOUS DIARRHEA IN CHILDREN

Two Caused by B. dysenteriae (Sonne) and One by B. dispar

Marion M. Johnston, Ph.D., and Mildred J. Kaake
Toronto, Ontario

I. B. DISPAR INFECTIONS

FIFTEEN cases of infectious diarrhea comprised a small epidemic among newborn infants in the nursery of a small maternity hospital in Toronto. Nine babies died.

About May 12, 1933, two breast-fed infants who apparently had been progressing satisfactorily developed loose stools which numbered from four to six per day. The diarrhea was accompanied by vomiting. These symptoms persisted for a week when the attending physician advised that they be removed to the Hospital for Sick Children for more extensive treatment. On May 19 when they were admitted to this hospital, their temperatures were 98° F. The examinations of their upper respiratory systems showed no involvement of the noses or ears; their throats were slightly red. On May 20 paracentesis was performed on one child, and pus was obtained; this child died on May 23. The second baby survived until June 4; otitis media developed about May 22. No other complications in their clinical history were noted. No macroscopic blood or pus was observed in the stools of these babies.

The other babies who developed similar clinical symptoms following these two had not been born when the two first babies were removed to this hospital. On May 31 one case was admitted to the Hospital for Sick Children; on June 2, four cases; on June 3, two; on June 5, one; and on June 6, two. There were two later cases which were not transferred. No cultures from new cases were received after June 17. Cultures from a total of fourteen cases were received.

These babies varied in age from eight to seventeen days when admitted to the Hospital for Sick Children. Two had been discharged from the maternity hospital for two days before admission to this hospital. During the two-day interval one vomited after each feeding; the other was well for one day, after which vomiting and diarrhea began and continued for two days until the child was brought to this

From the Research Laboratories of the Department of Pediatrics, University of Toronto, and the Hospital for Sick Children, under the direction of Alan Brown, M.D., F.R.C.P. (C)

hospital. In regard to the other cases, symptoms had persisted for from two to seven days prior to admission.

Stools varied in number from four to ten per day; they were light yellow in color and watery in consistency. No macroscopic blood or pus was observed at any time.

Bacteriologic studies of stool, nose, and throat showed that the only uncommon species of microorganism which occurred frequently was *B. dispar*.[1] This bacterial species was isolated from the stools of eleven babies, and from the nose and throat of one additional baby in whose stool we failed to find it. At postmortem it was also isolated from colon, middle ears, and brain of one child; from duodenum, small intestine, and colon of a second; from middle ears, small intestine, and colon of a third. It was recovered from the catheter urine specimen of a child whose feces had also contained the species. It was cultured during life from the noses and throats of four babies, from three of whose stools it was also derived. One blood culture taken postmortem was negative. No other bacterial species of importance were noted in nose and throat cultures.

The cultural characteristics of *B. dispar* are as follows: It is a gram-negative bacillus which is nonmotile, does not liquefy gelatin, or reduce lead acetate. It acidifies and coagulates litmus milk and produces indol. No gas was demonstrated in a peptone water medium containing 1 per cent dextrose. Acid is produced in peptone water medium containing Andrade's indicator and separately 1 per cent of the following carbohydrates, lactose, dextrose, maltose, saccharose, xylose, mannite.

On June 10, serum from five babies was tested for agglutinins with homologous strains as antigens. All were negative. On June 25, three of the five were retested and again were negative.

Later stool cultures from survivors showed one child positive.

In a search for the source of the epidemic, stool cultures were carried out on the five nurses who were in any way concerned with the nursery. On June 8 the stool of one of the five yielded *B. dispar*. Three subsequent samples obtained from the same nurse were negative. No agglutinins for her strain were demonstrated in her serum.

From the stool cultures of twelve healthy babies carried out after the epidemic had subsided, one strain of *B. dispar* was obtained.

In reviewing previous bacteriologic studies of cases among children presenting similar clinical symptoms, *B. dispar* was obtained from the stools of thirty-five out of 353 children (infants under two and older children), all of whom had diarrhea and enteritis. No other species of pathogenic significance was isolated from these cases. It was not obtained from thirty-one normal children, the studies being carried out at the same seasonal period. *B. dispar* was isolated from the urine of eighteen out of 315 pyelitis cases.

PATHOGENICITY OF B. DISPAR

B. dispar was isolated and described by Passet in 1885 from a rectal abscess. It seems to have been isolated from various sources since then. In 1907 Castellani[1] described a bacillus apparently similar to *B. dispar* which he recovered from the blood of a man in Ceylon ill with diarrhea for about two months. The patient's serum agglutinated his homologous strain in a dilution of 1:300. It was pathogenic by intraperitoneal injection for guinea pigs and rabbits, producing death in from twenty to thirty-six hours. Topley and Wilson[2] described *B. dispar* as "appearing to be a cause of bacillary dysentery in man."

W. Leonard Forsyth, department of bacteriology, University of Cairo, has recently reviewed the data with respect to *B. dispar*. He says: "It is probably a commensal which becomes a concomitant when the normal flora of the bowel is disturbed by the entry of a bowel pathogen. It cannot be accepted as a dysenterogenic organism until further serologic evidence is obtained from the study of many strains."

Castellani obtained one positive serum. In the present epidemic none have been found positive. However, negative findings have been the result in Sonne dysentery infections when the child was not progressing and later died.

II. B. DYSENTERIAE (SONNE) INFECTION

Outbreaks occasioned by *B. dysenteriae* (Sonne) have been reported by Cann and De Navasquez,[4] MacGill and Downie,[5] Smith,[6] Leahy,[7] Felsen and Osofsky.[8]

In November, 1931, a small epidemic of diarrhea involving sixteen cases was reported from a residential institution in this city. The first stool specimens in four cases were received on November 16; one, on November 18; three, on November 19; one, on November 20; two on November 24; one, on November 25; three, on December 2; one, on December 7. *B. dysenteriae* (Sonne) was isolated in eight cases. Of those cases in which stools yielded this species, it was obtained from the first specimen submitted in six cases, not until the second specimen was examined in one, and in the fourth in one. The organism persisted from November 16 to December 2 in one case in which six specimens were submitted.

Most of these children, under four years of age, were not acutely ill. They had slightly increased temperatures. Stools varied from two or three to six per day. Blood was noted in stools of two. One child suffered more severely. She passed as many as ten stools per day with some blood in two. Her temperature rose to 104° F. She was transferred to the Hospital for Sick Children where she was a patient for nine days before readmission to the institution from which she had come.

Four nurse contacts were examined in an effort to trace the source of this outbreak. The stool cultures from all were negative. Two specimens from each of three were cultured. The source of the infection was not determined. All the children made uneventful recoveries.

III. B. DYSENTERIAE (SONNE) INFECTION

Early in September, 1934, an outbreak of a small epidemic involving five children and one nurse occurred in a surgical ward in this hospital. All patients recovered. On August 31 a two-year-old boy was admitted to the ward with a tentative diagnosis of head injury following a fall about eighteen hours prior to admission. A few hours after the fall he vomited and had abdominal pain. There was no diarrhea. After admission his temperature was 102° F., and he passed green stools with blood and mucus. He was toxic and dehydrated. No stool cultures were made until September 12, when *B. dysenteriae* (Sonne) was isolated. He never had diarrhea, about two stools per day being the average.

Following the admission of this child, four additional cases developed in patients in adjoining beds in the same ward. The first case developed on September 1, a child, aged two and three-fourths years, who had been in the hospital since June. She developed loose stools with mucus but no blood or pus. Her temperature rose to 103.8° F., and on September 5 *B. dysenteriae* (Sonne) was isolated.

The next new case was observed on September 4 when the child, a boy four and one-half years of age, admitted on August 24, had a temperature elevated to 102° F. and passed loose green stools showing a few pus cells. The Sonne bacillus was isolated.

The third case was diagnosed on September 7 when a three-and-one-half-year-old boy with a temperature of 103° F. passed watery greenish stools. *B. dysenteriae* (Sonne) was cultivated from a stool submitted that day.

The fourth case did not develop until about September 9, when a slight increase in temperature was noted in a boy aged two years and two months, admitted with a diagnosis of appendicitis. There was no diarrhea or vomiting, and no blood, mucus, or pus was noted in his stools. The first culture was negative, but a second sent for examination on September 12 was positive.

During the course of this epidemic a nurse who had been in charge of this group was indisposed for one day. She was nauseated in the morning and had diarrhea but no other symptoms. She was able to continue her duties and was entirely recovered the following day. No stool specimen was submitted for examination, but it is probable she had the same infection.

It appears that the first patient experienced no ill effects from his fall and that he was incubating the Sonne bacillus which caused his pain and brought about the vomiting and his increased temperature. Until the stools containing blood were passed, when a diagnosis of dysentery was made, he was retained in a ward where he evidently was the source of infection for other cases. The exact means of conveyance of infection was not determined.

SUMMARY

1. An outbreak of diarrhea apparently due to *B. dispar* involving fifteen infants in the nursery of a small hospital is reported. Nine deaths occurred.

2. Two epidemics apparently caused by *B. dysenteriae* (Sonne) are reported. One outbreak in a residential institution involved sixteen patients; the other occurring in a surgical ward of this hospital affected five patients and one nurse. The infections were not acute and terminated quickly without fatalities.

REFERENCES

1. Castellani: J. Hyg. 7: 1, 1907.
2. Topley and Wilson: Principles of Bacteriology and Immunity, Vol. 1, p. 446.
3. Forsyth, W. L.: J. Trop. Med. 36: 65, 1933.
4. Cann, L. W., and De Navasquez: J. Hyg. 31: 361, 1931.
5. MacGill, J. S., and Downie, A. W.: Lancet 2: 29, 1932.
6. Smith, R. E.: Lancet 2: 925, 1931.
7. Leahy, A. D.: Am. J. Pub. Health 21: 1126, 1931.
8. Felsen, J., and Osofsky, A. G.: J. A. M. A. 103: 966, 1934.

THE TONSIL AND ADENOID PROBLEM

The Systemic Influence of Upper Respiratory Infection

George Morrison Coates, M.D., and William Gordon, M.D.
Philadelphia, Pa.

INTEREST in this subject has been recently revived by a number of excellent symposiums conducted here and abroad.

In 1931 J. M. Le Mée, of Paris, presented his very thorough study of this question, and in 1933, under the auspices of such distingiushed laryngologists as Herbert Tilley, J. A. Glover, Dan McKenzie, and others, the Fellowship of Medicine in London took up certain phases of this subject in the form of a "debate." It was discussed in great detail by Lee W. Dean at the 1934 meeting of the American Medical Association, by Kaiser, Nissen and Mosher at the recent American Academy meeting in Chicago, by the writers in May, 1934, at a symposium before the Philadelphia County Medical Society, and by the American Academy of Pediatrics, Region II, at San Antonio, Texas, Nov. 14, 1934, Mitchell, Dean, Shea, McCulloch, and McLaurin participating.

Many years ago John Nolan MacKenzie, of Baltimore, startled the profession with his "Massacre of the Tonsils," while the London thesis was "that operations for removal of tonsils are too often performed without adequate cause."

In order to discuss these questions in a logical and rational manner, let us review very briefly the clinical anatomy, histopathology, and clinical pathology of the lymphoid structures in the pharynx in general, and the tonsils and adenoids in particular. Following this, we shall discuss the physiology and function of these structures, the relationship of tonsillar sepsis to organic disease elsewhere, and what constitutes a proper diagnosis of tonsillar infection. Finally, the entire theme will conclude with a few concepts, which, it is hoped, will lead to a closer understanding, and therefore a fuller cooperation, between the internist, pediatrician, and the otolaryngologist.

The faucial tonsils, two in number, consist in the main of a fibrous capsule from which trabeculae pass into the parenchyma of the tonsil. These trabeculae carry blood vessels, nerves, and *efferent* lymphatics, i. e., lymphatic channels away from the tonsil, there being *no afferent lymphatics.* The parenchyma of the tonsil consists of the germinating follicles and the interfollicular tissue, all of which are located between

Read before the combined meeting of the Section on Otolaryngology of the College of Physicians of Philadelphia and the Philadelphia Pediatric Society, Dec. 11, 1934.

the trabeculae. The free surface of the tonsil presents ingrowths of surface epithelium of the stratified squamous type. The epithelium dips in to form the crypts. Lymphoid cells infiltrate the cryptal epithelium to such an extent that there may be, and usually are, two or three layers of epithelium separating the lumen of the crypt from the tonsil parenchyma.

Grossly the faucial, or palatine, tonsils are two more or less almond-shaped masses of adenoid tissue placed one on each side of the oropharynx and between the pillars of the fauces. In childhood they are more globular but recede as age advances. On the free or mesial surface, there are present a number of pits or crypts varying in depth from 1 mm. to 1 cm. or even more, the larger crypts often expanding below the orifice so that retention of large quantities of secretion or débris can take place. Not infrequently the mouths of these crypts become sealed by reason of acute or chronic inflammation, and the sac then has no outlet and may take on some of the characteristics of an acute or chronic abscess. No mucus glands open into the bottoms of these crypts. The crypts of the lingual tonsil are short and wide, and their openings are funnel shaped. They do not easily harbor débris, as pointed out by Dean.

The pharyngeal tonsil, or adenoid, often spoken of as the third or Luschka's tonsil, occupies the vault of the pharynx, at times almost completely filling this cavity.

With the growth of the individual, the tendency of the adenoid to atrophy is more marked than in the case of the faucial tonsil, and, indeed, it is comparatively rare to find any great amount remaining after adolescence. The adenoid is a lobulated organ having a succession of grooves and ridges ranging in number from three to five. While focal infection is often attributed to the faucial tonsils, the adenoid as a focus is commonly overlooked, although it may be the true source of the disease. This is because the adenoid is out of sight behind the velum of the palate and because, in adults, it is assumed that it has undergone atrophy.

Dean tells us that the pharyngeal tonsil and the infratonsillar nodules, i. e., the lymphoid structures at the base of the tongue in the laryngopharynx, contain crypts which become more readily impacted with débris than those of the lingual tonsils and less easily impacted than those of the faucial tonsils.

Histopathologic examination of these structures presents a variety of cellular elements, notably plasma cells, eosinophiles, and polynuclear leucocytes in the tonsillar parenchyma and in the crypts. So much for the anatomic and histopathologic considerations.

The physiology and function of the tonsils and lymphoid structures in the pharynx has long been a moot question. Some physiologists

and pathologists say that the tonsils possess a defensive mechanism against respiratory infection though clinicians on a series of controlled studies were unable to confirm this. Many believe that the cytogenic function of the tonsil is the most plausible one. This theory suggests that the healthy tonsil helps to confer immunity by protecting against bacterial invasion. Microanatomic studies in the laboratory have shown that white corpuscles can migrate from the lymphoid tissue of the tonsils into the crypts between the stratified squamous epithelium, gaining entrance into the pharynx where they destroy microorganisms. Pilot, Davis, and others are not in accord with this idea.

Though many may differ in minor details regarding the probable method of how tonsils help in immunity, almost everyone who has given careful study to this subject agrees in principle that the healthy tonsil does help in immunity, particularly during the early years of life, and that under six years of age it has a protective function. It is then the first line of defense, while the cervical glands are the second line of defense. Histologically lymphocytes may be observed migrating through the cryptal epithelium into the crypts, returning into the lymph nodules where the bacteria are destroyed.

It has been taught for a good many years that the crypts are virtual test tubes where microorganisms multiply and elaborate toxins. These toxins are absorbed with resultant autoimmunization. Waldapfel believes they are concerned in filtering out particulate matter from the blood stream. It has been shown that they produce lymphocytes, that they react against stimulants, bacteria, and any toxin which may penetrate into the lymphoid tissue. It is known that they develop after birth and continue to function but should involute at puberty. Hormone production, internal secretions, digestive ferments, blood forming, and protection for the lower respiratory tract are among some of the functions ascribed to these structures by many, though some of these theories have more adherents than others.

In connection with the foregoing it is well to state at this point that we fully appreciate the fact that a healthy tonsil does have one or more functions and that these are most active during the early years of life.

Our next consideration will be as to what constitutes a proper diagnosis of tonsillar infection. This is obtained by a careful history and examination of the lymphoid elements in the pharynx.

The history of local tonsillar inflammation, acute or chronic, regardless of the organisms present, or repeated attacks of tonsillitis (Hajek's dictum) is absolute evidence of tonsillar infection. One or more attacks of quinsy, repeated coryzas beginning with sore throat, soreness and discomfort in the tonsillar region, enlargement or tenderness of the node at the angle of the jaw (G. B. Wood), cervical adenopathy otherwise unaccountable, catarrhal or suppurative otitis media, rheu-

matic or arthritic manifestations, endocarditis, nephritis, pyelitis, lower respiratory infection, ocular infections, or other distant symptoms require a most careful investigation of the lymphoid structures in the pharynx.

Simple inspection of the fauces will usually fail to determine with any accuracy whether or not a tonsil is infected, and it may be stated here that an absolutely negative report is never possible until the tonsil has been removed and studied histologically and bacteriologically. Small, sealed crypts deep in the tonsil substance are not uncommon and are often impossible of detection even with the transillumination method of French. A careful inspection of the fauces will often reveal a purplish red tinge limited to the anterior pillar which is supposed to have some pathologic significance, especially where the infecting organism is a streptococcus. Often masses of cheesy débris may be observed filling the crypts if the anterior pillar is withdrawn by a pillar retractor. If, in addition, pressure is made external to the meridian of the tonsil, cheesy masses or liquid grayish or yellowish pus or mucopus will flow from one or more crypts, the superior ones in the upper pole being those in which this secretion is most apt to accumulate. Adenoid examination is less frequently made, but where undertaken, after removal, the presence of large numbers of polynuclear leucocytes is reported by the pathologist.

As long as the body resistance and the immunologic powers of the individual are able to cope with the bacteria and their toxins elaborated in the crypts and the parenchyma of the lymphoid structures, no deleterious systemic lesions are produced. As soon as these vital powers are diminished, however, the bacteria and their toxins are enabled to attack important organs and may set up irreparable pathologic changes.

The toxins may enter the parenchyma from the crypts and either be absorbed by the venous capillaries or be removed by the efferent lymphatics. Soluble toxins may be absorbed directly from the tonsil into the blood stream, thus accounting for the early intoxication in acute tonsillitis. Infection may also spread along the perivascular lymph spaces, sometimes contrary to the direction of the blood stream (Dean).

It is not generally appreciated that the adenoid may have just as serious focal possibilities as the tonsils, teeth, or sinuses. Bacterial multiplication readily takes place in the deep crypts, in which, after removal, pus is often noted. From this source infection may travel down the nasopharynx to the oropharynx and laryngopharynx and to the tracheobronchial tree. Cultures taken from the crypts of adenoid masses show growth in 100 per cent of cases. The adenoid, in simple hypertrophy, is of greater significance than the tonsil because of its location, for it may impair nasal respiration.

It may not be amiss, at this point, to outline very briefly some of the conclusions of the Le Mée study conducted a few years ago, reference to which has been made at the beginning of this paper. J. M. Le Mée sent out a questionnaire on this subject to clinicians all over the world. The referendum was not addressed to any particular group but to internists, pediatricians, orthopedic and general surgeons, opthalmologists, otolaryngologists, and pathologists. Nearly every medical center in the world was represented. The replies of the respondents, several hundred in number, carefully tabulated and printed in book form, are of interest as presenting a cross-section of world opinion. Here are a few of the most apropos questions and a summary of the answers to them.

Question: "Do you believe that the tonsils may constitute a portal of entry and a focus for secondary infections?"

Affirmative answers were almost unanimous.

Question: "What place do you assign to the tonsils among generally accepted foci, namely, the teeth, ears, gallbladder, and prostate?"

Answer: In four classes:

Class I (60 per cent of the replies) assigned first place to the tonsils. Class II (18 per cent) assigned second place to the tonsils, giving first place to the teeth. Class III (12 per cent) held that the tonsils shared equally with other foci. Class IV (10 per cent) assigned first place to the tonsils in children and first place to the teeth in adults.

Question: "Relation between the tonsils and various forms of rheumatism (acute, chronic, and deformans)?"

Answer: I. Affirmative replies, 90 per cent.

A. Almost unanimous for acute rheumatism.

B. For a relation to chronic rheumatic infections, 45 per cent. Improvement here is very frequent after tonsillectomy.

C. Rheumatism (arthritis) deformans, 5 per cent. Tonsillectomy is almost never followed by improvement.

II. Doubtful, 10 per cent.

To the question, "On what signs do you base your opinion that the tonsils are septic and may be a cause of secondary infection?" the collective answer is as follows:

1. Escape of pus from the crypts spontaneously or after pressure on the anterior pillar (50 per cent of the replies).

2. Repeated exacerbations of acute tonsillitis or peritonsillar abscess (65 per cent).

3. Congestion of the anterior pillars (50 per cent).

4. Cervical adenopathy (40 per cent).

5. Presence or retention of caseous matter (20 per cent). In the opinion of those who gave this answer the caseum unaccompanied by pus suffices to characterize the tonsil as a "bad" or "responsible" one.

6. Buried, small, or "adherent" tonsils (20 per cent). (George B. Wood protests against the term "adherent." What is meant is that by the deposit of fibrous tissue around the capsule, due to chronic or repeated acute infections, the tonsil is more or less immobilized and can be dislocated from its fossa with great difficulty.) The buried, small, or adherent tonsil is particularly suspected by English and American writers.

7. Simple tonsillar hypertrophy with enlargement of the cryptal orifices and eventually the presence of small ulcero-congestive lesions at their borders (10 per cent).

8. Bacterial examination (5 per cent), especially if streptococcus is recovered. Judson Daland has often spoken and written advocating this method of diagnosis. His dependence is placed upon the type of organism recovered and the number of colonies produced.

To make a diagnosis of tonsillar infection, then, we must make use of all the above mentioned points, but the clinical evidence should always be studied in its possible relationship to the history before the tonsil is condemned—that is, if we wish to be conservative, as, we believe, we should.

That the tonsils often act as portals of entry for infection manifested in other parts of the body is generally accepted, as well as that chronic disease of the tonsils may be a primary cause of infectious processes of focal origin elsewhere in the body. It is often overlooked that the infected tonsil may be only *one* of several such agencies, although perhaps the primary one. Pemberton called attention to the fact that even though focal infection was responsible for many kinds of disease, many individuals get well even *in the presence of surgical foci of infection*. This led to a series of detailed studies that were very revealing. Metabolic studies, glucose tolerance tests, blood chemistry, blood gas studies, studies of the blood vascular system, blood flow, sweat, body mechanics, and elimination were undertaken. He believed that each case had to be studied thoroughly and that there existed no panacea for rheumatism. His conclusion was that, in addition to the removal of foci of infection, all measures calculated to normalize metabolism, to promote blood flow, to dilate the peripheral vessels, to minimize toxemias, to lessen the body load, and to promote elimination should be used in a properly studied and treated case.

While the tonsils may be proved to be, beyond question, infected, we are unable thus far to say with certainty whether or not they are the absolute, or primary, cause of the systemic disease in any given case. There may be multiple causes present, and they should all be dealt with as thoroughly as possible. The infected tonsil may cause or perpetuate another focus which may be directly responsible, as for example one in the gastrointestinal tract. Other foci of infection,

therefore, should not be overlooked when considering a tonsillectomy because of a diagnosis of septic tonsils. Such foci may well be in the teeth, sinuses, possibly the ears, gallbladder, gastrointestinal tract, and prostate.

However that may be, whether the tonsils and adenoids are responsible in whole or in part for the systemic infection, or whether they have no relationship to it whatsoever; once the fact that they are definitely infected is established, they should be removed.

We do not wish to go on record as believing that every tonsil is infected, but we cannot with any assurance say that a given tonsil is not infected unless that tonsil is removed and subjected to a careful bacteriologic and pathologic examination. How often have we removed a fairly innocuous appearing tonsil, only to see it gush pus from every crypt when squeezed by the snare or guillotine? To certify that any given tonsil is definitely not a focus is to court diagnostic disaster.

From a conservative standpoint, before tonsillectomy is performed, in consideration of systemic infection, it must be decided whether the tonsillar infection is probably actually causing damage or is likely to cause it in the future. Regarding prophylaxis, there is great divergence of opinion.

Kaiser, in a ten-year study of the school children of Rochester, N. Y., seems to show statistically that prophylactic tonsillectomy did not make for great immunity to recurrent infections of the upper respiratory tract, to acute contagious disease or to rheumatism, and indeed, that lower respiratory infections occurred with greater frequency in tonsillectomized children than in those in whom the tonsils were not removed. The studies of Horace Williams in the Philadelphia Hospital for Contagious Diseases, over a similar period, showed rather conclusively that the children in this hospital who had middle ear suppuration and its complications were almost all not tonsillectomized.

Dean, in commenting on Kaiser's report of the increased incidence of laryngitis, bronchitis, and pneumonia in tonsillectomized children, believes that this is due to the fact that at the time of the tonsil operation, nasal sinusitis was present and that this nasal sinusitis is responsible for the pulmonary disease. Further, he believes that most of the nasal sinus conditions can be controlled by dietetic and hygienic treatment following the tonsil and adenoid operation.

Dan McKenzie doubts the value of statistics used to disprove the efficacy of a well-performed tonsil operation and asks whether the figures are reliable, whether in fact, in those cases in which focal disease has developed after tonsillectomy, we have any statistics to prove that the tonsils *were* completely removed, or whether the ade-

noids were not alone removed in some cases, and whether a recurrence of this lymphoid tissue was excluded by competent examination.

Herbert Tilley deprecates the wholesale removal of the tonsils as a prophylactic measure but believes that a definite septic tonsil should be removed. His method of diagnosis of a septic tonsil is substantially as outlined above, and above all he urges a careful study of the whole physical condition of the patient.

The diagnosis of tonsillar sepsis in relation to focal infection requires the most cooperative study. If it is a question of middle ear or sinus disease, the otolaryngologist may well decide upon the proper procedure. When, however, the case is one of arthritis, let us say, other problems arise, and the case should have the most careful study by all the physicians associated with the case. It will not do for the laryngologist who finds the infected tonsils to order immediate removal on the chance that they are the sole, or even the main cause, of the arthritis. Other foci must be sought, or causes other than infection, and the diagnosis of tonsil infection as the etiologic factor in the case must be arrived at by general consultation and consideration of all the evidence. Pemberton has brought this out very clearly, and he never allows a tonsillectomy, or any other surgical removal of a focus of infection, not only until the patient has been thoroughly studied, but also until he has been put in such physical condition that the operation will, at least, do no harm.

Kaiser well says that the reaction against indiscriminate tonsillectomy followed the less striking beneficial results when the tonsils were removed for vague indications, while statistical and clinical study justifies enthusiasm for this procedure in properly selected children.

Nissen, in his very excellent presentation of the subject, insists that after infection in the tonsils is *determined*, the study must be extended to all other parts of the body to demonstrate systemic disorder. Depending on the type of case, this may necessitate appropriate blood and urine studies, metabolic and endocrine tests, careful investigation of the cardiovascular system, respiratory tract, neurologic, gastrointestinal, kidney function, bones, joints, body mechanics, etc. If then, the systemic disorder is discovered, the relationship between the tonsillar infection and the systemic disorder must be proved. This will require close clinical observation by the physician and frequent reports of subjective symptoms by the patient over a considerable period of time.

From the laryngologic standpoint alone, certain principles that were written elsewhere may be emphasized here. Every part of the pharynx and fauces should be examined with care. It is not enough to know that a tonsil operation has been performed. The fossae must be examined for stumps, tags, regrowths, small crypts concealed behind the

pillars or beneath scar tissue, compsensatory hypertrophies of the lateral pharyngeal folds, and isolated lymphatic masses in the posterior pharyngeal wall, infected adenoid remnants, infected lymphoid structures at the base of the tongue (the infratonsillar nodules in the laryngopharynx), whose importance has been emphasized by French, as well as for the gravitation of infected secretions from an infection of the posterior series of sinuses or an extension of an inflamed mucosa.

CONCLUSIONS

1. The ultimate diagnosis of infection of the tonsils, adenoids, and the other lymphoid structures of the pharynx rests with the laryngologist.

2. By painstaking examination, either the presence or probable absence of sepsis will, therefore, be determined.

3. If infection is demonstrated, then a prophylactic clean-up of the lymphoid elements of the pharynx may give striking results.

4. No tonsil and adenoid operation should ever be performed after a hasty and inadequate examination.

5. When a septic tonsil is discovered in association with some systemic disorder, the diagnosis of tonsillar infection as the cause of this disorder can only be satisfactorily arrived at after meticulous study and consultation, often repeated if necessary, on the part of the laryngologist with the attending pediatrician, internist, or surgeon.

POISONING FROM THE BITE OF THE BLACK WIDOW SPIDER (LATRODECTUS MACTANS)

CASE REPORT

E. H. ROHLFING, M.D., AND B. M. BULLINGTON
ST. LOUIS, MO.

ALTHOUGH a number of cases of poisoning from the bite of the black widow spider have been reported in the United States,[1] we feel that this condition has not received sufficient emphasis as the clinical picture may be easily confused with an acute abdominal lesion. The mental symptoms may be of such a nature[2] that it is difficult to obtain an accurate history. This latter phase is particularly true in children when the knowledge of the bite may not be known to the parents. Because of this it is important that patients with the symptoms of an acute surgical abdomen be carefully questioned as to the possibility of being bitten.

W. M., a white male three years old, was seen (by E. H. R.) at 4:00 P.M. and immediately referred to St. Louis Children's Hospital. The history was as follows: About five hours previously the child was bitten by an insect which the parents killed and found to be a "black spider which had a red belly." The child was bitten as he carried a sprinkling can in the yard around the flower beds. The bite produced considerable discomfort, and soon an area of redness appeared around the site of the injury. There were no other complaints at this time, and the child pursued his usual activities until about one and one-half hours later, when he suddenly began to complain of a severe, constant, agonizing pain in his abdomen which soon "doubled him up." There was no nausea, vomiting, convulsions, or fever. The mother gave the child an enema, which was expelled clear and afforded no relief. Since the onset of the abdominal pain there had been little change in the boy's condition.

The family and past histories were not significant, except that the child had been in "good health" until his present illness. Physical examination showed a three-year-old boy, lying in bed, acutely ill, with his knees and thighs flexed on his abdomen. He was quite restless and complained of a severe constant pain throughout his abdomen and a milder discomfort in his right forearm. On the dorsal surface of the right forearm there was an erythematous, raised area about the size of a silver dollar. There was no lymphadenopathy nor evidence of lymphangitis.

His temperature was 37° C.; respirations, 44 per minute; pulse, 120 per minute.

The examination of the head, eyes, ears, nose, and mouth showed nothing abnormal except moderately dilated pupils, which reacted to light and accommodation. His respiratory movements were rapid, entirely thoracic, shallow, accompanied by an expiratory grunt, but without dilatation of the alae nasi. The chest was clear. The heart was not enlarged; the sounds were of good quality, regular in rate and rhythm. The abdomen was flat and possessed a boardlike rigidity throughout. Palpation produced no additional discomfort, and because

of the rigidity none of the viscera could be palpated. No free fluid could be detected. The reflexes were normal except for the presence of slightly hyperactive knee jerks and biceps. No Babinski, Chaddock, Oppenheim, Gordon, Hoffmann, Kernig, or Brudzinski reflex was elicited. There was no neck nor back rigidity detected.

Laboratory Data.—Red blood cells 4,900,000; hemoglobin (Sahli), 90 per cent; White blood cells, 19,000. Differential count—basophiles, 0; eosinophiles, 0; lymphoblasts, 0; myeloblasts, 0; myelocytes, 0; juveniles, 0; stabs, 14; segmenteds, 64; lymphocytes, 18; monocytes, 4. No parasites nor punctate basophilia was seen.

Urine: The reaction was alkaline to litmus; sugar, 0; albumin, 0; ketone bodies, 0; specific gravity, 1025, microscopic examination revealed an occasional white blood cell, no red blood cell, occasional epithelial cells, and no casts.

Temperature ranged from 37 to 37.5° C.

The treatment in this case was magnesium sulphate as used by De Asis,[4] except that we used the intramuscular route. Ten cubic centimeters of a 25 per cent solution of magnesium sulphate was given intramuscularly in the gluteal regions. Elixir of sodium phenobarbital was given in amounts of 4 c.c. every four hours for three doses. Following this medication, the child rested fitfully through that night. The next morning he was physically improved; he had less pain and a fair appetite, but some abdominal rigidity was still present. His recovery was rapid, and he was discharged from the hospital after twenty-four hours. At the end of forty-eight hours the abdominal rigidity had entirely disappeared, and the child was quite well again. At the time of discharge his white count was 17,600 with a differential count of stabs, 7; segmenteds, 55; lymphocytes, 32; monocytes, 6.

This case represents a rather typical course of poisoning by a black widow spider bite. The problem of diagnosis would have been exceedingly difficult had not there been the specific history obtainable from alert parents. The possibility of confusing the picture with that of an acute surgical abdominal lesion is shown when the features of the clinical picture are reviewed.

REFERENCES

1. Bogen, E.: Arch. Int. Med. **38**: 623, 1926.
2. Walsh and Morgan: Am. J. M. Sc. **186**: 413, 1933.
3. Morton: Arch. Surg. **26**: 64, 1933.
4. De Asis, Cesareo: Am. J. Trop. Med. **14**: 33, 1934.

3720 WASHINGTON BOULEVARD

VITAMIN A AND INFECTION

ISAAC F. GITTLEMAN, M.D., AND ALEXANDER S. WIENER, M.D.
BROOKLYN, N. Y.

THE relation of vitamin A to infection has been the subject of numerous studies. All investigators agree that gross vitamin A deficiency in animals and humans is associated with increased susceptibility to spontaneous infections, which may become evident before other external signs of deficiency make their appearance. This increased susceptibility has been ascribed[1-5] to a certain characteristic change in the epithelium of the bronchopulmonary, genitourinary, and alimentary tracts. This consists in the replacement of the normal epithelial lining by stratified keratinized epithelium, which is thought to be less impregnable to invading bacteria than is the normal lining membrane.

Because vitamin A deficiency disease in animals and humans was corrected or, if not too far advanced, was cured by supplying an adequate amount of this vitamin in the food, some workers believed that vitamin A might exert an antiinfective or protective action.

With this assumption clinical studies were made in which vitamin A was used as a therapeutic agent during the course of acute infections. Mellanby and Green[6] reported in 1929 that they had obtained beneficial results by treating cases of puerperal sepsis with a vitamin A concentrate. However, these experiments were not well controlled, and the series of cases treated was small. Subsequent investigators failed to corroborate their claims.[7] At Mellanby's suggestion, Orenstein[8] studied the effect of vitamin A concentrate in the treatment of pneumonia in mine workers since it was believed that their diet might be deficient in this vitamin. A series of 764 cases was divided at random into two equal groups, only one of which received the treatment. This study revealed that the extra vitamin A had no effect on the mortality rate, length of stay in the hospital, or number of complications.

A clinical study of the effect of vitamin A on the complications and mortality in measles was carried out by Ellison[9] on a series of 600 patients, 300 of whom were given supplementary vitamin A and D. He found the mortality in the treated group to be 3.7 per cent (eleven deaths) in contrast to a mortality of 8.7 per cent (26 deaths) in the untreated group. Strangely enough, however, the incidence of com-

From the Department of Pediatrics, the Jewish Hospital of Brooklyn, and the Brooklyn Hebrew Orphan Asylum.

plications, such as pneumonia and otitis media, was practically identical in the two groups. Of the thirty-four patients with pneumonia in the control group, twenty-three died, and of the thirty-two patients with pneumonia in the especially treated group, ten died, so that practically all the deaths were due to pneumonia. This study, therefore, reduces itself to an analysis of the effect of vitamin A (and D) on the mortality from pneumonia. Ellison's small series of pneumonia cases was apparently not as well controlled as Orenstein's large series since in the former study the control group was kept in a different ward from the specially treated group, so that there was a possibility of differences in hygienic and nursing care.

A similar study has been made by Sutliff, Place, and Segool[10] on the effect of vitamin A on the incidence of otitis media as a complication of scarlet fever. They found that the supplementary feeding of vitamin A had no beneficial effect. On the whole, the above investigations seem to indicate that extra vitamin A has no effect on the course or mortality from infections, once they are established.

In spite of the fact that definite forms of vitamin A deficiency like xerophthalmia are believed to be rare at the present time, the latent form of this disease may be widespread, in view of the effect of unemployment on the diet of children in poor families. For this reason, a number of investigators have suggested the use of vitamin A as a prophylactic agent against deficiency and to reduce the incidence of infections. With this idea in mind, Green, Pindar, Davis, and Mellanby[11] followed two groups of 275 pregnant women, one of which groups was given a vitamin A and D concentrate. According to these investigators, the general morbidity was significantly higher in the untreated than in the treated group.

A number of investigations have been made to determine whether or not vitamin A has any prophylactic value against respiratory infections in children. In a study of sixty infants, Wright, Frosst, Puschel, and Lawrence[12] found no reduction in the incidence of respiratory infections when vitamin A concentrate was administered in addition to the cod liver oil given to all infants as a routine measure. Barenberg and Lewis[13] studied three groups of infants, one receiving only skimmed milk, which is presumably low in vitamin A content, the second receiving supplementary vitamin D, and the third receiving vitamin A and D combined. In this study also, no difference in the incidence of respiratory infection was found. In a more recent investigation by Gardner and Gardner[14] sixty-nine school children were followed over a three-month period. Twenty-two were used as controls; nineteen were given foods rich in vitamin A; and twenty-eight were given halibut liver oil. According to the authors, despite the fact that from

the previous history the children in the control group were more resistent to colds, these children had as many colds and missed more days of school during the experimental period than children in either of the other two groups. However, the differences found were small, and the data were given in a form which does not permit of statistical interpretation.

Probably the most extensive and best controlled investigation on this question has been conducted by Hess, Lewis, and Barenberg.[15] One group of seventy-seven infants was given a vitamin A supplement, and a second group of seventy-five infants was used as a control. Since in the former there were 245 respiratory infections during the experimental period which lasted five months, and in the latter, 241 infections, no significant effect was obtained from the extra vitamin A. In a similar study on a series of 118 cases, Mackay[16] failed to find any reduction of respiratory infections as a result of vitamin A feeding. Mackay also recorded the occurrence of other infections besides those affecting the respiratory tract and found that the number of skin infections was significantly higher in the control group.

Therefore, aside from the work of Green and his collaborators, who observed a significantly lower incidence of puerperal morbidity in women receiving supplementary vitamin A and D during their pregnancies, and the work of Mackay, who found fewer skin infections among infants given vitamin A, there have been no convincing reports concerning the value of vitamin A as a prophylactic agent against infection.

In the hope of throwing further light on this problem, the present investigation was undertaken. This study was carried out at the Brooklyn Hebrew Orphan Asylum, an institution which cares for children between the ages of seven and seventeen years. The children were divided into three groups: the first consisting of ninety-one individuals, receiving halibut liver oil (henceforth designated as the "A" group); the second consisting of ninety-four children, receiving viosterol (to be designated as the "D" group); and the third consisting of ninety individuals, acting as a control (designated as the "C" group). The division into groups was performed at random so that the age and sex distribution did not differ significantly in the three groups (Tables I and II).

All the children were given the regular institution diet consisting of green vegetables and fresh fruit, meat and fish, bread, butter, and milk.

SAMPLE MENU

Breakfast	Oatmeal, bread, butter, cocoa, sugar
Lunch	"Hamburger" loaf, mashed potatoes, peas, carrots, apples, bread
Dinner	Baked beans, bread, butter, chocolate pudding, milk

Breakfast	Wheatena, bread, butter, cocoa, sugar
Lunch	Spaghetti, tomato sauce, bread, jam, milk, cookies
Dinner	Salmon salad, mashed potatoes, rolls, butter, milk, fresh fruit, cake, bread
Breakfast	Two boiled eggs, bread, butter, cocoa, sugar
Lunch	Pot cheese, sour cream, bread, jam, milk, fruit, jello
Dinner	Boiled beef, parsley, potatoes, string beans, bananas, bread

TABLE I

AGE DISTRIBUTION IN THE THREE GROUPS OF CHILDREN STUDIED

AGE	A GROUP	D GROUP	CONTROLS (C GROUP)
7	6	3	13
8-9	14	18	11
10-11	8	10	3
12	17	8	17
13	15	14	8
14	9	15	15
15	15	13	14
16	7	6	5
17	0	7	4
Totals	91	94	90

TABLE II

SEX DISTRIBUTION IN THE THREE GROUPS OF CHILDREN

SEX	A GROUP	D GROUP	C GROUP
Male	67	64	68
Female	24	30	22
Totals	91	94	90

The children of the C group received in addition one teaspoonful of corn oil* in one ounce of tomato juice at their noon meal since this oil is known to be practically lacking in vitamin content. The halibut liver oil was given to the children of the A group in the dosage of a half teaspoonful daily in one ounce of tomato juice. The halibut liver oil used contained 71,960 international or U.S.P. X (1934) vitamin A units and 837 international or U.S.P. X (1934) vitamin D units per gram. The individuals in the D group were given 15 drops of viosterol daily on a small piece of bread. (The preparation used, viosterol 250 D, contains 10,000 international vitamin D units per gram.) All three preparations were distributed and consumed under the personal supervision of one of the authors.

Administration of the vitamin-containing supplement to the diet was instituted on Nov. 1, 1932, and was discontinued on May 31, 1933. However, the observations were continued until February, 1934, in order to detect any latent or cumulative effect due to storage of vitamin

*The corn oil, the halibut liver oil, and the viosterol used in this investigation were supplied through the courtesy of Mead Johnson and Company, who also standardized these preparations for their vitamin content. The corn oil used was devoid of vitamin A, carotene, and vitamin D.

A in the body. Routine physical examinations including height and weight, condition of nose, throat, and eyes were carried out at monthly intervals. In addition, children with elevation of temperature or other evidence of respiratory infection were required to report to the infirmary where they were examined by one of us and the nature of the ailment recorded.

With regard to height and weight, all three groups of children behaved similarly. Thus, 80 per cent of the individuals in the A group gained weight during the experimental period, 78.3 per cent in the D group, and 78 per cent in the C group. With regard to height, similar observations were obtained.

The relative incidence of infections in the three groups of children is given in Tables III and IV. The infections were classified under four main headings: respiratory infections, skin infections, contagious diseases, and miscellaneous infections. Since the vast majority of the

TABLE III

INCIDENCE OF INFECTIONS DURING THE PERIOD OF SUPPLEMENTARY FEEDING OF VITAMINS

TYPE OF INFECTION	CHILDREN RECEIVING HALIBUT LIVER OIL (A GROUP)		CHILDREN RECEIVING VIOSTEROL (D GROUP)		CONTROL GROUP (C GROUP)	
	NO. OF CHILDREN	NO. OF INFECTIONS	NO. OF CHILDREN	NO. OF INFECTIONS	NO. OF CHILDREN	NO. OF INFECTIONS
Respiratory (including complications)	24	30	27	35	32	38
Skin	5	5	0	0	3	3
Contagion	4	4	5	5	7	7
Miscellaneous	7	7	4	4	6	6
Totals§	35	46	35	44	41	54
Total number of children in each group	90		91		94	
Percentage of children with respiratory infections	26.7 ± 3.14*		29.7 ± 3.23*		34.0 ± 3.30*	
C-A†			7.3 ± 4.55*			
C-D‡			4.3 ± 4.61*			

*Probable error.

†Difference between percentage of children with respiratory infections in C Group and A Group.

‡Difference between percentage of children with respiratory infections in C Group and D Group.

§Totals of columns headed "No. of children" is less than the sum of the numbers of children with respiratory infections, skin infections, etc., since a number of children had more than one kind of infection.

illnesses were respiratory in nature, the statistical analysis is restricted to this type of infection. During the experimental period, the A group had the smallest number of respiratory infections and the C group, the largest. The differences found were within the limits of statistical error, however, as is shown in Table III. How the figures given in the table were derived may be illustrated by the following example.

The formula for the probable error of a frequency is: P.E. $= 0.6745\sqrt{\frac{p(1-p)}{N}}$, where p is the observed frequency and N the number of cases.

In the A group, for example, $p = \frac{24}{90} = 0.267$, or 26.7 per cent, and $N = 90$.

Therefore, the probable error $= 0.6745\sqrt{\frac{(0.267)\ (0.733)}{90}} = 0.0314$, or 3.14 per cent.

In a similar way the other frequencies and their probable errors were calculated.

To obtain the probable error of the differences, the following formula is used:

$$\text{P.E. difference} = \sqrt{(\text{P.E.}_1)^2 + (\text{P.E.}_2)^2}$$

Thus, the difference between the percentage of children with respiratory infections in the C group and A group is 34.0 – 26.7 = 7.3 per cent, and the probable error of this difference is

$\sqrt{(0.0314)^2 + (0.0330)^2} = 0.0455$, or 4.55 per cent.

Since none of the differences obtained are greater than three times the probable error, they cannot be considered significant, statistically.

After the discontinuation of vitamin feeding, the differences in the incidence of infection in the three groups observed were even smaller (Table IV). Since these differences were obviously not significant, no special statistical analysis is made.

TABLE IV

INCIDENCE OF INFECTIONS AFTER DISCONTINUATION OF VITAMIN FEEDING

TYPE OF INFECTION	CHILDREN RECEIVING HALIBUT LIVER OIL (A GROUP)		CHILDREN RECEIVING VIOSTEROL (D GROUP)		CONTROL GROUP (C GROUP)	
	NO. OF CHILDREN	NO. OF INFECTIONS	NO. OF CHILDREN	NO. OF INFECTIONS	NO. OF CHILDREN	NO. OF INFECTIONS
Respiratory	21	25	15	20	17	21
Skin	0	0	1	1	0	0
Contagious	2	2	0	0	3	3
Miscellaneous	1	1	4	4	4	4
Totals	23	28	20	25	21	28

In the further pursuit of this study an attempt was made to determine what the incidence of respiratory infection in these three groups of children was before the experimental diet was initiated. The only source of information which could be consulted was the medical charts of the children. There was no way of determining whether or not all the illnesses had been recorded as faithfully previously as during the experimental period. However, if there had been any omissions, these omissions must have affected all three groups equally, so that the data could be used for determining the *relative* incidence of infections in the three groups. The incidence of recorded infections during the fifteen-month period before the experiment was instituted is given in Table V. As was the case during the experimental period, the smallest number of infections was found to be in the A group and the largest in the C group. Surprisingly enough,

however, the differences here are decidedly significant statistically. The cause for the difference could not be determined.

The last observation serves to illustrate one of the errors an investigator may fall into when interpreting the results of a statistical study. Had the differences during the experimental period been significant, statistically, the tendency would have been to attribute them to the effect of the supplementary vitamin in the diet. The data in Table V demonstrates, however, that there may be other factors beyond the investigator's control which affect the results. This seems to have been the case, for example, in Ellison's study of the effect of vitamin A feeding on the mortality from measles.

TABLE V

RELATIVE INCIDENCE OF INFECTIONS IN THE THREE GROUPS OF CHILDREN BEFORE EXPERIMENTAL PERIOD

TYPE OF INFECTION	A GROUP		D GROUP		C GROUP	
	NO. OF CHILDREN	NO. OF INFECTIONS	NO. OF CHILDREN	NO. OF INFECTIONS	NO. OF CHILDREN	NO. OF INFECTIONS
Respiratory infections	7	9	20	21	27	30
Skin infections	4	4	2	2	9	9
Contagion	3	4	0	0	7	7
Miscellaneous	2	2	3	3	6	6
Totals	16	19	24	26	49	52
Total number of children in each group	90		91		94	
Percentage of children with respiratory infections	7.69 ± 1.89		21.28 ± 2.85		30.0 ± 3.26	
C-A	22.3 ± 3.8					
C-D	8.7 ± 3.4					

As has already been pointed out, it is now generally accepted that in cases of marked vitamin A deficiency, the susceptibility to infection is increased. It has been found that the vitamin deficiency and the concomitant susceptibility to infection can be overcome by feeding relatively small quantities of the vitamin, although the exact amount required is unknown. The negative results obtained in the present study and also in the studies of most other investigators, are possibly attributable to the fact that the control group was already receiving an adequate amount of vitamin in the diet, so that the addition of an excess of vitamin A failed to produce any further beneficial effect. On the other hand, the fact that some investigators, such as Mackay, and Green, and her collaborators, obtained positive results may be due to the particular types of individuals they studied, namely, people from the poorer classes who probably could not afford to purchase the essential components of a well-balanced diet.

From the evidence presented in this paper it seems clear that the usual "normal" dietary is adequate from the standpoint of its content

of vitamin A. The incidence of respiratory infections in children receiving such a regimen is therefore dependent on factors other than the dietary one investigated. It must not be concluded, however, that latent forms of vitamin A deficiency, which are easily overlooked, do not occur. Indeed, recent pathologic and clinical studies of Blackfan and Wolbach[5] and others[16, 17] have proved the existence of this malady in malnourished children. Furthermore, among children who for one reason or another are placed by the physician on restricted diets, the occurrence of vitamin A deficiency is a distinct possibility. In such instances supplementary vitamin A in the diet may be essential.

SUMMARY

Three groups of children whose ages varied from seven to seventeen years were studied. One group received supplementary halibut liver oil in the diet; the second, viosterol; and the third group served as a control. No statistically significant difference in the incidence of respiratory infections was found.

The authors wish to take this opportunity to express their appreciation and indebtedness to Mr. Aaron Jacobi, Director, and the entire staff of the Brooklyn Hebrew Orphan Asylum without whose assistance this investigation would have been impossible.

REFERENCES

1. Wolbach, S. B., and Howe, P. R.: J. Exper. Med. **42**: 753, 1925.
2. Wolbach, S. B., and Howe, P. R.: Arch. Path. & Lab. Med. **5**: 239, 1928.
3. Wilson, J. R., and Dubois, R. O.: Am. J. Dis. Child. **26**: 431, 1923.
4. Wolbach, S. B., and Howe, P. R.: J. Exper. Med. **57**: 511, 1933.
5. Blackfan, K. D., and Wolbach, S. B.: J. Pediat. **3**: 679, 1933.
6. Mellanby, E., and Green, H. N.: Brit. M. J. **1**: 984, 1929.
7. Burton, A. H. G., and Balmain, A. R.: Lancet **1**: 1063, 1930
   Cramer, W.: Lancet **1**: 1153, 1930.
8. Orenstein, A. J.: South African M. J. **6**: 685, 1932.
9. Ellison, J. B.: Brit. M. J. **2**: 708, 1932.
10. Sutliff, W. D., Place, E. H., and Segool, S. H.: J. A. M. A. **100**: 725, 1933.
11. Green, H. N., Pindar, D., Davis, G., and Mellanby, E.: Brit. M. J. **2**: 595, 1931.
12. Wright, H. P., Frosst, J. B., Puschel, F., and Lawrence, M. R.: Canad. M. A. J. **25**: 412, 1931.
13. Barenberg, L. H., and Lewis, J. M.: J. A. M. A. **98**: 199, 1932.
14. Gardner, E. L., and Gardner, F. W.: Am. J. Dis. Child. **47**: 1261, 1934.
15. Hess, A. F., Lewis, J. M., and Barenberg, L. H.: J. A. M. A. **101**: 657, 1933.
16. Mackay, H. M. M.: Arch. Dis. Child. **9**: 65, 1934; **9**: 133, 1934.
17. Jeans, P. C., and Zentmire, Z.: J. A. M. A. **102**: 892, 1934.

555 Prospect Place

ENDOCRINE STUDIES IN INFANTS AND CHILDREN

II. Time of Onset of Teething, Walking, and Talking in Endocrine Disorders: Its Relationship to Intelligence in Endocrine and Nonendocrine Conditions

Murray B. Gordon, M.D., and Lawrence Kuskin, M.D.
Brooklyn, N. Y.

THE relative lack of the statistical approach to the study of endocrine conditions in children prompted us to analyze 1,000 cases at our disposal from the viewpoints of developmental rate, mental status, clinical findings, chemistry of blood and urine, metabolism, and x-ray studies of the bones. These various phases will be discussed in separate papers. The methods of procedure and the diagnostic criteria of endocrine conditions have already been described.

The present paper reports the findings of a statistical study of the time of onset of teething, walking, and talking. In 570 patients the case history presented definite data on at least two of the following developmental criteria: the age at which the first deciduous tooth was erupted, the age at which unaided walking began, and the age at which the first words of the child's vocabulary were spoken. We are assuming the validity of evidence secured by good case histories obtained from the average mother.

The 570 children are distributed as follows: 369 present evidence of some endocrine disorder, and 201 manifest some degree of mental retardation without any evidence of endocrine disturbance. The endocrine group is in turn almost equally subdivided into those with normal intelligence and those with mental retardation. The clinical entities represented in the endocrine group are childhood myxedema (which we prefer to the term sporadic cretinism), hypothyroidism, adiposogenital dystrophy in boys, thyropituitary obesity in girls, and anterior pituitary deficiency of growth. In the nonendocrine mental retardation are included those due to mongolism, encephalitis, congenital syphilis, spastic paraplegia, birth injury, head injury in infancy or childhood, epilepsy, and an unclassified group.

The mental status was determined by direct evidence on the basis of psychometric examinations or by indirect evidence as presented by case histories, parental information, and reports from public and parochial schools in those instances where the children were of school age.

From the Department of Pediatrics, Long Island College of Medicine, and the Endocrine Clinics of the Long Island College Hospital and Israel Zion Hospital.

TABLE I

TIME OF ONSET OF TEETHING, WALKING, AND TALKING IN ENDOCRINE CONDITIONS

ENDOCRINE CONDITION	NO. OF CASES	TEETHING		WALKING		TALKING	
		M.* IN MO.	S. D.† IN MO.	M. IN MO.	S. D. IN MO.	M. IN MO.	S. D. IN MO.
Childhood Myxedema	27	14.89	7.07	29.04	8.05	33.92	16.51
Hypothyroidism							
All cases	47	9.68	5.05	20.20	8.31	25.73	12.25
Normal mentality	10	7.67	2.52	17.0	4.69	19.62	9.27
Retarded mentality	37	10.31	6.23	20.29	7.89	24.16	11.84
Adiposogenital Dystrophy							
All cases	137	8.35	3.47	15.14	5.01	18.13	10.70
Normal mentality	89	7.85	2.62	13.98	2.73	15.60	5.81
Retarded mentality	48	9.43	4.62	17.17	8.10	23.74	15.56
Thyropituitary Obesity							
All cases	116	8.03	2.89	15.57	5.92	16.0	7.59
Normal mentality	73	8.33	2.73	13.52	3.72	14.57	8.45
Retarded mentality	43	7.68	2.99	18.67	7.86	18.94	8.26
Anterior Pituitary Deficiency							
All cases	42	11.05	5.05	16.4	5.3	17.42	7.26
Normal mentality	34	9.30	2.39	15.06	4.59	16.97	7.17
Retarded mentality	8	17.62	7.21	22.71	8.31	20.4	5.39

*M.—Mean average.
†S. D.—Standard deviation.

The incidence of mental retardation in each endocrine group is as follows: childhood myxedema, 97.5 per cent; hypothyroidism, 89.3 per cent; adiposogenital dystrophy, 41 per cent; thyropituitary obesity (female), 36 per cent; and anterior pituitary deficiency, 19 per cent.

TABLE II

TIME OF ONSET OF TEETHING, WALKING, AND TALKING IN NONENDOCRINE MENTAL RETARDATION

CONDITION	NO. OF CASES	TEETHING		WALKING		TALKING	
		M.* IN MO.	S. D.† IN MO.	M. IN MO.	S. D. IN MO.	M. IN MO.	S. D. IN MO.
Mongolism	53	15.96	6.22	29.32	10.37	30.78	14.27
Postencephalitis	26	8.38	3.25	21.79	7.56	24.59	12.82
Congenital syphilis	17	12.8	6.4	21.05	10.03	21.73	9.25
Spastic paraplegia	38	9.71	3.82	38.12	20.73	31.65	13.71
Head injury	7	7.71	1.27	16.66	4.13	21.6	6.1
Epilepsy	6	8.4	1.85	16.0	2.4	28.66	12.77
Unclassified mental retardation	55	9.32	3.62	21.04	9.28	34.85	14.27

*M.—Mean average.
†S.D.—Standard deviation.

The time of onset of teething, walking, and talking in children with normal intelligence in the endocrine group is compared with that in mentally retarded children in the endocrine and nonendocrine groups. We did not study the question of sex relationship.

FINDINGS

Because of the apparent relationship between the mental status and the time of onset of teething, walking, and talking, it was decided that all analyses take into consideration the intelligence of the child.

Teething.—The average age of eruption of the first deciduous tooth is generally accepted to be between six and nine months in normal children. Table I presents the average (mean) and standard deviation obtained in each entity studied in this series arranged according to the mentality. Our report is based upon these figures and upon additional technical studies, which need not be discussed in detail.

In the mentally normal endocrine group we find that dentition is delayed in anterior pituitary deficiency and is normal in hypothyroidism, adiposogenital dystrophy, and thyropituitary obesity. There seems to be no significant difference in the teething time in the latter three groups, especially between the last two if we compare normal with normal children and retarded with retarded children. Premature teething, that is prior to the sixth month, is noted only in the pituitary disorders, occurring in 29.7 per cent in adiposogenital dystrophy, in 23.2 per cent in thyropituitary obesity, and 14.3 per cent in anterior pituitary deficiency of growth.

The relationship of mental status to teething is shown in an analysis of the endocrine group on this basis irrespective of the disease entity.

Children with normal mentality show an average teething time of 8.53 months (S.D., 2.71), while those with mental retardation show an average of 10.36 months (S.D., 6.13). This indicates that mentally normal children with endocrine conditions teethe at an earlier average age than mentally retarded children with endocrine disorders. In the individual clinical entities of the endocrine group, we note that the difference in teething time between the normal and mentally retarded is 8.32 months in anterior pituitary deficiency, 2.64 months in hypothyroidism, and 1.58 months in the adiposogenital dystrophy group. In female thyropituitary obesity there is a normal history of teething irrespective of the mental status. In childhood myxedema the average age of teething is 14.89 months. Every child with this latter condition had an intelligence quotient of 75 or less except one girl who tested 81.

In the mentally retarded endocrine group, children with myxedema teethe later than those with adiposogenital dystrophy, female thyropituitary obesity, and hypothyroidism but earlier than those with anterior pituitary deficiency. There is no evidence in our series that this latter difference is due to anything but chance factors. With the exception of the last group, we can state that children with myxedema will teethe later than those with any other endocrine disorder irrespective of the mental status.

Infants with nonendocrine mental retardation due to antenatal conditions such as mongolism, microcephalus, and congenital syphilis teethe later than those with postnatal disorders like epilepsy, spastic paraplegia, birth injury, brain trauma during infancy, and encephalitis. It is interesting to note that in our so-called "unclassified mental retardation group" the average time of onset of teething is below nine months. This is probably due to the heterogenity of the group which contains instances of both antenatal and postnatal disorders and is therefore not truly representative of either.

Walking.—The average age for walking without aid in normal nonendocrine children is considered to be fifteen months. In our series the mentally normal children in the endocrine group walked at an average age of 14.34 months (S.D., 3.68), while those mentally retarded in the same group on the average did not walk until 19.11 months (S.D., 7.74 months). Calculation of the probable error of the difference between the two subgroups indicates that normal children will walk earlier than those mentally retarded.

In mentally normal infants the onset of walking appears at about the same time in all endocrine conditions, with a slight delay in the cases of hypothyroidism and anterior pituitary deficiency. The latter may be due to chance factors. In mentally retarded children in the endocrine group, the delay in walking is greatest in the order named: childhood myxedema, anterior pituitary deficiency, and hypothyroidism.

Walking is delayed to a greater extent in all types of nonendocrine mental retardation. The greatest delay is found in mongolism and to a lesser degree in those types which developed in the postnatal period, like postencephalitis, brain injury, and epilepsy. Spastic paraplegia cannot be included in this analysis because of the involvement of the muscular system.

Talking.—The average age for the onset of talking in normal infants is between twelve and sixteen months. In our series, this is within normal limits in all mentally normal infants with pituitary conditions, except for a slight delay, which may be due to chance factors, in anterior pituitary deficiency. There is also a slight delay in mentally normal infants with hypothyroidism.

It is practically certain that infants with mental retardation will talk later than normal infants if all the endocrinopathies are grouped together as one. Delay in talking is greatest in mentally retarded infants in the order named: childhood myxedema, hypothyroidism, adiposogenital dystrophy, anterior pituitary deficiency, and thyropituitary obesity. It is probable that the difference between the last two groups are due to chance. It is interesting that infants with anterior pituitary deficiency and mental retardation do not show comparatively the same degree of delay in talking as in teething.

In the entities studied, children with nonendocrine mental retardation due to antenatal factors talk later than the mentally retarded in the endocrine group.

DISCUSSION

Any study of endocrine influence upon the developmental rate must take cognizance of possible nonendocrine factors. In the consideration of the latter, racial stock, heredity, and familial tendencies play important rôles. Colored races are more precocious in teething time than the white. Economic conditions seem to have some effect as shown by the delay in eruption in the poorer class of children in orphan asylums.

Conclusions as to the relationship between the time of onset of teething and the general stature are not conclusive. Some claim that, everything else being equal, taller children will teethe earlier than short children; others limit this relationship to those of the same racial stock and economic condition. Acute infections and chronic conditions like rickets have an inhibiting effect on the eruption of the first teeth.

A number of determinants have been described in walking. The development of the spine with its changing curvature, the shifting of the center of gravity in the developing infant, and the growth of muscle tissue and bone have all been stressed as important factors in the development of walking. Size, racial stock, sex, and presence of acute infections may also be determining factors.

The endocrine factor in developmental growth has been recognized. Engelbach states that the thyroid hormone influences tissue differentiation, intrauterine growth, the laying down of embryonal tooth buds, and the subsequent eruption of the teeth. Hoskins and others have called attention to the relationship of the thyroid hormone to the mental status and the integrity of the central nervous system. On the basis of our findings in this series we may go further and state that there seems to be a relationship between the mental status, thyroid activity, and the time of onset of teething, walking, and talking. We do not know at present whether this triple relationship depends upon the activity of the thyroid hormone alone or upon a common third factor.

The effect of thyroid deficiency on the three criteria seems to bear a relation to the time of onset of both the endocrine disorder and the mental disturbance.

Intrauterine thyroid deficiency as exemplified by childhood myxedema and congenital hypothyroidism will in all probabilities cause early mental retardation and delay in teething, walking, and talking. Infantile hypothyroidism or myxedema will affect each of the three criteria depending upon the chronologic relationship between the onset of the disorder and the normal time for teething, walking, and talking. If this deficiency occurs prior to the age of six to nine months, all three criteria will be affected; if later than six to nine months teething time will be normal and walking and talking, delayed. In our series the onset of hypothyroidism in the mentally normal group probably occurred after the normal time for teething and before that for walking and talking. The results may not be valid because of other chance factors in the small number of cases analyzed.

The rôle of the pituitary gland in teething is not as well known as that of the thyroid. Engelbach states that pituitarism, without the designation of hypoactivity or hyperactivity, is characterized by early eruption, that is, prior to the sixth month. The tendency in anterior pituitary deficiency of growth is not stated.

Our findings indicate that the anterior pituitary lobe plays an important rôle in the time of appearance of the deciduous teeth and that the latter is linked up with growth processes in general. In mentally normal children with pituitary obesity (adiposogenital dystrophy and thyropituitary obesity) and normal or increased stature, the onset of teething is normal, and in 22.4 per cent of the cases, premature. On the other hand, in children with anterior pituitary deficiency of growth and normal mentality, the delay in teething time is more marked than in any other endocrine class with normal mentality. This delay may be part of the general slowing up of all growth processes in this condition. It may be an endocrine explanation of the observation that

taller children of the same racial stock teethe earlier than short children. We cannot state whether or not there exists any great difference between the various racial stocks in our series. (The races represented are those of cosmopolitan New York.) It is quite probable that anterior pituitary deficiency of growth produces a delay in teething irrespective of the racial stock. It is difficult to evaluate the parts played by the growth hormone itself and by the thyrotropic hormone.

The relation between the onset of teething and mental retardation is definitely shown in our series. There is a statistically reliable difference between children of normal and retarded intelligence, but there is no exact mathematical relationship between the time of teething and the degree of intelligence.

It does not necessarily follow that a markedly retarded child will teethe later than one mildly retarded, but it implies that a mentally retarded child will teethe later on the average than one mentally normal with the same endocrine condition. This relationship of intelligence to teething has been noted by Terman and others. The presence or absence of endocrine conditions in their series was not stated by them.

Our findings that mentally retarded children with endocrine conditions will talk and walk later than mentally normal children with the same disorders corroborates the observations of Abt and coworkers. These investigators studied the relationship between intelligence and the onset of speech and of walking in 1,000 children with an intelligence quotient ranging from 10 to 159. The presence or absence of endocrine disorders was not stated. They found a definite relationship between intelligence and talking and that the higher the intelligence quotient, the earlier the child learns to talk. They also concluded that the higher the intelligence, the earlier the child begins to walk.

The etiologic relationship between endocrine condition, mental retardation, and the onset of teething, walking, and talking may be briefly summarized as follows: 1. They may be all expressions of some common factor present during antenatal or neonatal life. 2. The glandular disturbance may be the cause of the delayed developmental rate and/or the mental retardation. 3. The delay in teething, walking, and talking may be due to the mental retardation irrespective of the existing endocrine condition.

An evaluation of these relationships is especially difficult when the mental retardation is associated with an endocrine disorder which may of itself cause delayed dentition (as in childhood myxedema, congenital hypothyroidism, or anterior pituitary deficiency) or delayed talking (as in childhood myxedema or congenital hypothyroidism).

An added difficulty in interpretation is encountered when the mental retardation itself may be due to endocrine disorder as in childhood myxedema or congenital hypothyroidism. A neoplasm, inflammation, or injury in the pituitary region may be the common factor in the production of mental retardation, pituitary disturbance, and delay in developmental growth.

On the basis of our findings in this series, it is difficult for us to evaluate these various factors.

SUMMARY AND CONCLUSIONS

A statistical study is presented of the time of onset of teething, walking, and talking in endocrine disturbances and of the relationship of intelligence to the developmental rate. Of 570 children studied, 369 presented evidence of endocrine disturbance, and 201 manifested some degree of mental retardation without any sign of endocrine dyscrasia. The endocrine group is almost evenly divided into those with normal intelligence and those with mental retardation. The developmental rate in mentally normal children in the endocrine group is compared with that in mentally retarded children in this group and in children with mental retardation associated with nonendocrine conditions.

As far as we can ascertain, there are no statistical studies of a large series on the time of onset of teething, walking, and talking in endocrine conditions. We, therefore, cannot compare the averages and standard deviations obtained in this study with those of any other investigator.

The findings in our study may be summarized as follows:

1. The time of onset of teething, walking, and talking depends upon four factors: (A) the functional activity of the thyroid gland and of the growth hormone of the anterior pituitary lobe; (B) nonendocrine factors such as racial stock, familial tendencies, chronic conditions (rickets, etc.), acute infections, etc.; (C) the mentality of the infant in the first six months of life; (D) the chronologic relationship between time of inception of the influencing factor (endocrine or nonendocrine) and the normal time of teething, walking, and talking.

2. In a mentally normal child, the time of onset of teething, walking, and talking may serve as a criterion of endocrine function. Delay in the appearance of one or all indicates endocrine disturbance, provided all possible nonendocrine factors are excluded.

3. In a mentally retarded child, the time of onset of teething, walking, and talking cannot be considered as a diagnostic criterion of endocrine disorder.

4. Teething is within normal age limits in mentally normal children with adiposogenital dystrophy, thyropituitary obesity, and hypo-

thyroidism. In the pituitary group there seems to be a relationship between the time of onset of teething and general growth. Teething time is normal with normal functioning of the growth hormone but is delayed with deficiency of the latter. Dentition is delayed to the greatest extent in anterior pituitary deficiency of growth and in childhood myxedema. Premature teething is noted in 22.4 per cent of the pituitary cases in this series and is not noted in any other glandular disturbance. The time of eruption in hypothyroidism depends upon the relationship between time of inception of the dyscrasia and normal teething time and also upon the mentality of the infant in the first six months of life.

5. Unaided walking appears in mentally normal infants at about the same average time in all endocrine conditions except hypothyroidism in which there is a slight delay. In mentally retarded children, the delay is greatest in the order named: childhood myxedema, anterior pituitary deficiency, and hypothyroidism.

6. The onset of talking is within normal limits in mentally normal children in all pituitary conditions and is slightly delayed in hypothyroidism. In mentally retarded infants, talking is delayed to the greatest extent in the order named: childhood myxedema, hypothyroidism, adiposogenital dystrophy, anterior pituitary deficiency and thyropituitary obesity.

7. Infants with mental retardation teethe, walk, and talk later than mentally normal infants in the same endocrine groups. Those with mental retardation due to nonendocrine antenatal factors such as mongolism, congenital syphilis, and microcephalus teethe, walk, and talk later on the average than those with postnatal disorders such as epilepsy, encephalitis, or brain injury. Considered as two groups, children with nonendocrine mental retardation in this series teethe, walk, and talk later than those with endocrine disorders.

8. The relationship between endocrine conditions, mental retardation, and delay in onset of teething, walking, and talking may be as follows: A. They all may be expressions of some common factor present during antenatal or neonatal life. B. The glandular disturbance may be the cause of the delayed developmental rate and/or the mental retardation. C. The delay may be due to the mental retardation irrespective of the existing endocrine disorder. On the basis of our findings in this series, it is difficult for us to evaluate these possible factors.

We are indebted to Mr. Bernard Berkowitz for his technical assistance.

REFERENCES

Abt, I., Adler, H. M., and Bartelme, P.: The Relationship Between the Onset of Speech and Intelligence, J. A. M. A. 93: 1351, 1929.

Boas, F.: The Eruption of Deciduous Teeth Among Hebrew Infants, J. Dent. Research 7: 245, 1927.

—: Report of an Anthropometric Investigation of the Population of the United States, J. Am. Statistical A. 18: 181, 1922.
Boas, F., and Wissler, C.: Statistics of Growth: Reports of the U. S. Commissioner of Education, 1904, p. 25.
Cattell, P.: Dentition as a Measure of Maturity: Harvard Monographs in Education, No. 9, Cambridge, 1928, Harvard University Press.
Channing, W., and Wissler, C.: The Hard Palate in Normal and Feeble-minded Individuals: Anthropological Papers of the American Museum of Natural History, vol. 1, publication 5, p. 283.
Engelbach, W.: Endocrine Medicine, Springfield, Ill., 1932, Charles C. Thomas, vol. 2, p. 454.
Fenton, J.: A Practical Psychology of Babyhood, Boston, 1925, Houghton Mifflin Company.
Gebhart, J. C.: The Growth and Development of Italian Children in New York City: Publications of the Association for the Improving of the Condition of the Poor, 1924, publication 132.
Gordon, M. B.: Endocrine Studies in Infants and Children: I. Methods of Procedure and Diagnostic Criteria, J. PEDIAT. 5: 659, 1934.
Halverson, H. M.: An Experimental Study of Prehension in Infants by Means of Systematic Cinema Records, Genetic Psychol. Monogr. 10: 107, 1931.
Hellmann, M.: Changes in Human Face Brought About by Development, Internat. J. Orth. & Oral Surg. 6: 13, 1927.
Hellmann, M.: In Growth and Development of the Child, Part 2, White House Conference on Child Health and Protection, New York, 1933, Century Co., p. 149.
Hoskins, R. G.: Neuro Endocrinology, Chapter in Cyclopedia of Medicine, vol. 5, Philadelphia, 1932, F. A. Davis, p. 169.
Krogman, W. M.: Anthropological Aspects of the Human Teeth and Dentition, J. Dent. Research 7: 1, 1927.
Schwartz, L.: Résumé, With Comments, of Available Literature Relating to Posture, Pub. Health Rep. 42: 1219, 1927.
Simpson, W. G.: A Chronicle of Infant Development, J. Ment. Sc. 39: 378, 1893.
Smith, S.: The Influence of Illness During the First Two Years of Infant Development, J. Genetic Psychol. 39: 284, 1931.
Smith, M. E., Lecker, G., Dunlap, J. W., and Cureton, E.: The Effects of Race, Sex and Environment on the Age at Which Children Walk, J. Genetic Psychol. 38: 489, 1930.
Spier, I.: Physiological Age: The Relationship of Dentition to Body Growth, Dent. Cosmos, 1918, p. 899.
Terman, L. M.: Genetic Studies of Genius. I. Mental and Physical Traits of a Thousand Gifted Children, Palo Alta, Calif., 1925, Stanford University Press.
Variot, G.: Bull. et mém. Soc. méd. d. hôp. de Paris 51: 353, 1927.
Variot, G., and Gotcu, P.: Bull. et mém. Soc. d'anthrop. de Paris 8: 17, 1927.

Critical Review

THE NEWBORN

BARNET E. BONAR, M.D.
SALT LAKE CITY, UTAH

IT IS most gratifying to observe the increasing interest in problems relating either directly or indirectly to the neonatal period, for, as has been pointed out frequently, such problems have not received the attention due them in the past. Even in danger of becoming tedious by repeatedly pointing to the lack of information about the newborn, one cannot help but continue to do so with the hope that the present interest will be maintained or increased. Now that there is greater concern about maternal and neonatal problems, certain difficulties present themselves, particularly in respect to the manner in which investigations along these lines shall be undertaken. Since maternal and neonatal subjects in general are so intimately linked with one another, both must be studied together. Therefore, it is necessary for the obstetrician and the pediatrist to join forces in order to avoid duplication of effort and possibly considerable confusion in respect to interpretation of results. So far this has not occurred to any great extent. Thus, both obstetric and pediatric organizations have committees working separately without intercourse with one another, each attempting to effect a reduction of maternal and neonatal mortality rates, respectively. One of these, the American Committee on Maternal Welfare, which is striving through state medical societies to organize as a nation-wide agency to foster the practice of better obstetrics throughout the country, has been greeted enthusiastically by the medical profession in most states. Another, the Committee on Neonatal Morbidity and Mortality of the American Pediatric Society, is attempting to correlate existing fundamental knowledge of the newborn so that the various problems needing investigation can be attacked more successfully. With such committees engaged in active work in their various fields, one cannot but hope that they will see their way clear to consolidate their attack by becoming interlinked in some way so that the information obtained by each may be used to greater advantage. The same may be said of other organizations formed for the same purpose, and especially is this true of some of those originating from lay groups. Certainly a problem of such scope as this one has turned out to be needs guidance which will utilize every possible short cut so that the most can be accomplished with the least effort. To do this, coordination is necessary and, above all, strict medical supervision. With the medical profession interested in the subject as it is today, there would seem to be little excuse, indeed, for these other groups to take other than a subordinate part.

In a former review[1] mention was made of the difficulty in deriving reliable information from available vital statistics, owing to variations in

the methods of collecting data. For instance, such basic terms as "live-birth," "stillbirth," "prematurity," "viability," and "abortion" are not interpreted the same in the several states or countries. As a result of this variability of definition, erroneous interpretations of statistics may result in the dissemination of unreliable information about maternal and neonatal subjects. A similar situation prevails in respect to the nomenclature used by the medical profession in making diagnosis of morbid states. Terms used by doctors of one school do not have exactly the same meaning in the eyes of doctors of another school, and not infrequently the terms used by either group do not conform to those listed in the International Classification of the Causes of Death. Very apparent, therefore, is the need for those engaged in maternal and neonatal studies to talk the same language. Otherwise, erroneous interpretations and judgments will continue to be made. At present the only solution seems to lie in the adoption of a temporary arbitrary nomenclature which can be interpreted the same way by all investigators. An example of this is the term "prematurity." Clinically it matters little by what yardstick one determines what constitutes prematurity, for after all the clinician desires only to know whether the development of a given infant is such that he needs especial care if he is to be given the best opportunity to live and thrive. However, to the investigator it is essential that some standard of prematurity be agreed upon so that the reports of different workers may be analyzed properly. For instance, if a report is made of the mortality rate in a group of premature infants cared for in a certain way, it is necessary for the critic to know what standard was used to determine maturity, in order to learn whether or not borderline cases in which the mortality rate is low anyway have been included. For that reason it would be wise to agree upon some arbitrary means by which to measure prematurity. Whether the standard adopted is 5 pounds or 2,500 grams matters little. Another need is the publication of all data from which conclusions are derived so that they may be scrutinized by others. Furthermore, the mathematical significance or reliability of the figures should be determined in all of these reports.

The examples mentioned above point out only a few of the many existing deficiencies in our present research methods, but they serve to emphasize how important is the need for standardizing, temporarily at least, certain methods of approach so that some of the fundamental principles of the subject may be more clearly understood. It is hoped, therefore, that the several agencies organized to undertake the study of maternal and neonatal problems for the purpose of reducing mortality rates may be prevailed upon to cooperate in some way so that a coordinated project may be evolved. It would, perhaps, pave the way for a similar plan in other fields of medicine where it is also greatly needed.

The purpose of a review of this kind is to report some of the recent contributions on certain subjects with the idea of indicating the trend of medical thought in some instances and pointing out the need for further study in other instances. Above all, an attempt should be made to outline a rational course for the practitioner to pursue in respect to problems about which there is a diversity of opinion. It is particularly with this latter thought in mind that a few more or less controversial subjects are dealt with at this time.

THYMUS GLAND

For years the thymus has been the object of a great deal of controversy, and even today it is but little understood. It is a cervicothoracic lymphoid structure of broad triangular shape before the onset of respiration, after which it becomes altered to an elongated triangular form. The fact that it shrinks rapidly in size during disease and inanition or following death has led to considerable diversity in the figures given by different observers for the normal weight. Because correct weights are best obtained in normal infants dying from accident, Hammar's figures based upon individuals dying suddenly are accepted by most authorities.[2, 3, 4] He considers 15.15 mg. the normal weight of the thymus for the full-term newborn infant. Individual variations, however, are so great that this information is not particularly valuable. The gland increases rapidly in size up to the end of the second year, doubling its birth size at this time and trebling it at eleven years.[5] Involution then begins, but even in old age some functioning parenchyma may remain.

Little is known about the physiology of the thymus gland. After years of study Hammar was unable to prove that it produced an internal secretion, and up to the present time no one else has been able to do so in the human, at least. Painstaking extirpation studies on dogs led Park and McClure[6] to conclude that the thymus was not essential for life, and, in general, most observers agree that thymectomy is not followed by detectable symptoms. In animal experimentation the thymus has been associated with growth and development, and lately considerable emphasis has been laid upon its effect on prenatal growth in rats.[7] One of the chief troubles in studies of this nature is the difficulty of translating to the human the results of animal experiments. The gland, however, is considered an important source of blood lymphocytes and possibly of eosinophiles. Even from a pathologic standpoint there has been considerable controversy, save possibly for the rather well-known postmortem softening of the medulla and the rare condition of thymic apoplexy in which there occurs an extensive hemorrhage into the gland.[8] Some discussion has arisen about the origin and nature of both the so-called Dubois' abscesses, which will be mentioned later, and the neoplasms, such as malignant epithelial thymoma,[9] and so far it has not been possible to describe a characteristic pathologic picture of the thymus diagnosed as hyperplastic. It is over the enlarged thymus gland and its association with hyperplasia of other lymphoid structures, the so-called syndrome of status thymicolymphaticus, as well as suprarenal vascular deficiency and sudden death, that there has been so much discussion.

Enlargement of the Thymus Gland.—Fortunately, during the neonatal period one is less concerned with status lymphaticus than with a syndrome consisting of thymic enlargement, respiratory stridor, and asphyxial attacks of varying degree. Possibly some of the diversity of opinion which has developed over the thymus has been due, as Capper and Schless[4] point out, to an attempt to link the syndrome of the enlarged thymus, which occurs in the neonatal period and is not associated particularly with any distinctive body constitution, with that of status lymphaticus in which the thymus enlargement is merely a manifestation of generalized lymphoid hyperplasia.

The symptoms sometimes ascribed to thymic enlargement are paroxysmal, dry, brassy cough, a clucking or cackling sound, intermittent inspiratory stridor, continuous or intermittent dyspnea, choking, retraction of the head and sometimes spells of cyanosis. There may be associated with these symptoms such evidences of high laryngeal obstruction as retraction at the suprasternal notch and epigastrium.

While it is not possible to state exactly what constitutes an enlarged thymus gland, owing to considerable variation in its size in different infants, the gland is known to exert pressure upon the thoracic structures, for in the liveborn infant its surfaces bear the impress of all the organs with which it is in contact.[10] In some cases the thymic substance may extend posteriorly thereby compressing the structures behind it. This is due either to an enlarged gland or to a narrow superior thoracic aperture which will not allow it to be forced into the cervical region by the expanding lungs. It is possible that failure to take into consideration this latter factor may explain the obstructive symptoms in cases where the thymus is not enlarged. Another factor about which little is known is the relation of vascular engorgement to transient enlargement of the thymus gland.[11] Hart[12] states that the blood vessels with which the gland is so richly supplied become readily engorged and often contribute to the enlargement of the gland. Pancoast and Pendergrass[13] state that in tracheal encroachment by the thymus the point of greatest danger is the narrow thoracic aperture which is bounded by rigid osseous structures and that the apex of the wedge formed by the predominant cervicothoracic infantile thymus is forced further upward into this aperture during expiration. Since a natural collapse occurs in the posterior tracheal wall where there are no cartilaginous rings at the end of expiration, the apex of the wedge of a large thymus may intensify this collapse. Other observers have demonstrated compression of the trachea by large thymuses in vivo and at autopsy.[14, 15] Pressure paralysis of the recurrent laryngeal nerve with the characteristic brassy cough has also been noted. Pressure upon the heart, great vessels, and even the vagi has been suggested as a cause of thymic death, but so far there has been little evidence to support this idea. The same may be said for the possibility of pressure on the veins of the neck disturbing the cerebral circulation and eliciting convulsions. Clinically the diagnosis of thymic enlargement has been based upon percussion, palpation of the suprasternal notch, and x-ray examination. The latter furnishes the only method that can be relied upon to any extent and even its reliability is open to question. In the first place, anteroposterior views of the mediastinum in large series of cases have shown that large thymic shadows (extending beyond the vertebral transverse processes or more than 3 cm. wide) may be present in the newborn in percentages varying from 5 to 30 when symptoms are absent[16, 17, 18, 19] and that symptoms may be present when such roentgenograms reveal no enlargement. In the second place, it has been shown that wide shadows are found in overnourished children, which observation agrees with the anatomic studies mentioned above. In the third place, a good deal depends upon the phase of the respiratory and cardiorespiratory cycle when the roentgenograms are taken. Thus Gerstenberger[20] emphasized the necessity of obtaining roentgenograms during both deep inspiration and expiration because of the variation in the width of the shadow at these times. Because of the difficulty in obtaining

such negatives, fluoroscopic examinations should be used to check the findings of the roentgenogram. It has been shown also that the size of the thymus shadow depends upon what the phase of the cardiorespiratory cycle is at the moment the roentgenogram is taken.[21] Furthermore, it is the opinion of most radiologists that lateral views are of utmost importance in determining the effect of the thymus upon the trachea, for thickness is even more important than breadth in the causation of symtoms. Some believe that a wide shadow is safer than a dense, narrow one. Pancoast and Pendergrass[13] state that the only reliable roentgenologic evidences of a potentially dangerous thymus are:

1. A lateral deviation of the trachea on a sagittal view during inspiration
2. Narrowing of the tracheal lumen at the thoracic inlet during inspiration
3. Too great collapse at the inlet during expiration
4. Buckling of the trachea especially during inspiration
5. Marked posterior displacement of the trachea

All patients in whom enlargement of the thymus gland is suspected should receive a preliminary fluoroscopic study in the sagittal and lateral directions, and this must include the entire respiratory tract.

The relation of other factors to the so-called enlarged thymus syndrome is somewhat confusing. Suprarenal deficiency seems to be productive among other things of a thymic enlargement, and thymus hyperplasia is also found with thyroid hypersecretion. Aldrich[22] has reported low blood sugar determinations in a group of infants showing symptoms which might be ascribed to thymic enlargement. He attributes hypocalcemia to suprarenal insufficiency. Other complicating factors must be mentioned. Collapse of the soft tissues of the larynx and pharynx, which is of importance only when the thymus is obstructive, may cause noisy respiration. In the presence of atelectasis and bronchitis, or drowned lung, which may be due to other factors, an enlarged thymus may interfere with proper aeration of the lungs until it is reduced in size.[13] Wasson[23] is of the opinion that thymic enlargement is a coincidental finding and that probably the primary cause of the stridor is an infection of the bronchi, trachea, larynx, and accessory sinuses and that treatment of the infection will relieve the respiratory stridor. Such considerations bring up the question of the part thymic enlargement plays when some other complicating condition exists. Mitchell[24] has answered this admirably by applying statistical analysis to the problem, thus bringing into clinical thymus studies for the first time accurate calculations of probabilities. The mathematical implications were that: (1) the symptoms of stridor, cyanosis, and convulsions were not inevitably associated with enlarged thymus; (2) the symptoms mentioned, however, were more likely to be associated with enlarged thymus than not; and (3) these symptoms were more likely to occur when complicating conditions were found than when an enlarged thymus alone was found. Another condition which has been commonly thought to be associated with enlargement of the thymus is pylorospasm. Mitchell found that mathematically there was no obvious association between the two conditions.

The diagnosis of thymic enlargement entails differentiating the various causes of respiratory stridor, and undoubtedly many times the

symptoms are due to causes other than thymic enlargement. Many authors are of the opinion that most of the cases diagnosed as thymic enlargement are due to other causes.[4, 5, 24, 25, 26] It must be remembered that the symptoms may be due to a combination of an enlarged thymus and some other condition. These may be divided into extrinsic and intrinsic causes. The former consist of asthma, atelectasis, congenital heart disease, tumors of the thymus, lingual thyroid, micrognathia, cysts at the base of the tongue, retropharyngeal abscess, enlarged mediastinal glands, and congenital flaccid tongue. Most of these are seen but rarely. Of the intrinsic causes, the most frequent one is congenital laryngeal stridor due to a relaxation or atony of the larynx,[29] which is more common than ordinarily supposed. Other causes are laryngeal anomalies such as subglottic diaphragm, laryngeal infections, bilateral abductor paralysis, cerebral hemorrhage, laryngospasm or tetany of the newborn, neoplasms of the larynx,[27, 28] papillomas, and angiomas,[30] congenital stenosis, and congenital webs of the larynx.

The existence of such a large number of possible causes of stridor, many of which are difficult to diagnose, makes it all the more important to utilize every possible means of making a diagnosis. Careful and thorough fluoroscopic examination of the entire respiratory tract, sagittal and lateral roentgenograms, and endoscopic examination should be resorted to whenever circumstances permit. When in the presence of symptoms of respiratory obstruction and in the absence of other pathology which cannot be corrected an enlarged thymus is found, roentgenotherapy is advisable. Roentgen ray treatment of the upper mediastinum of infants manifesting so-called "thymic symptoms" possibly may affect some of the adjacent structures, such as the thyroid and parathyroids, but so far little information about this is available. The effect of such therapy upon the child later in life has been speculated upon by many. A study of sixty-three children from three and one-half to eight years after x-ray treatment of the thymus was made by Barnes.[21] Although the dosage was considerably greater than that used at present, these children manifested no constant deviation from normal, either physically or mentally. Failure to obtain relief from symptoms in an apparently uncomplicated case or too prompt relief indicates an erroneous diagnosis. The fact remains, however, that in a goodly number of instances relief does occur. Whether or not this is actually the result of treatment cannot always be determined, for relief might have occurred spontaneously; or, as Wasson[23] has pointed out, other treatment may have produced the same results, for instance, rather empiric treatment directed toward the correction of respiratory infections.

Summary.—The tremendous amount of conflicting and sometimes unjustifiable data about the anatomy, pathology, and physiology of the thymus gland, together with the difficulty in determining what constitutes an enlargement of the thymus, makes it exceedingly hard for the clinician to determine the course to pursue when confronted with the so-called syndrome of thymic enlargement. Undoubtedly a goodly amount of the confusion has been brought about by attempting to link this syndrome with that of status lymphaticus. The author is inclined to agree with Capper and Schless[4] that the enlarged thymus syndrome, which is first observed in the neonatal period, bears little if any relationship to status lymphaticus. However, the factors responsible for the

greatest amount of controversy, in the writer's opinion have been the failure upon the part of investigators: (1) to appreciate the fact that the results of animal experiments cannot be applied arbitrarily to the human; (2) to realize that the glands during life and at death are not exactly comparable; (3) to stress the importance of the superior thoracic aperture during the respiratory act; (4) to investigate the importance of vascular engorgement in relation to transient enlargements of the gland; (5) to recognize the need for fluoroscopy and sagittal and lateral roentgenograms during known phases of the respiratory cycle; (6) to emphasize the necessity for endoscopic examinations; (7) to recognize the need for greater effort to eliminate other causal factors before making a diagnosis; and finally (8) to accept the view that possibly an enlarged thymus may aggravate symptoms when another pathologic condition exists. Undoubtedly, thymic enlargement sufficient to cause serious pressure symptoms exists, regardless of statements to the contrary, but the uncomplicated incidence must be small. Enlargement with symptoms in the presence of such complications as congenital heart disease, increased intracranial pressure, suprarenal hemorrhage, respiratory infections, and tracheolaryngeal abnormalities may be and probably is more common. The possibility of mild attacks due to transient enlargement is still an open question. The term "thymic asthma" is erroneous and should be discarded. Certainly in the face of all the recent discussion, one should search carefully for some other possible cause of stridor before using the term "thymic enlargement" as loosely as it has been done in the past. The doubt cast by some of these reports has served to stimulate greater caution in making a diagnosis. However, in spite of the dogmatic assertions of some that there is no such syndrome, the conservative practitioner faced with what appears to him, after utilizing all the diagnostic aids at his disposal, to be enlarged thymus syndrome will probably feel safer in subjecting the infant to roentgenotherapy. For the time being, one might advantageously, at least from a clinical and therapeutic standpoint, differentiate between the symptomatic and nonsymptomatic enlarged thymus.

STATUS LYMPHATICUS*

In considering the thymus of the newborn, mention must be made of the so-called syndrome, "status lymphaticus," which, if it exists, must be latent during fetal and neonatal life. Status lymphaticus appears to be a constitutional or structural anomaly characterized by prominence of such lymphoid structures as the spleen, lymph nodes, intestinal follicles, tonsils, and thymus, and what is described as hypoplasia of the cardiovascular and chromaffin systems (suprarenal cortex and interstitial cells of the gonads). As can be seen from this, the thymus probably plays only a minor rôle. Considerable confusion has been brought about by the attempt of some to emphasize the importance of the thymus in the production of this syndrome, which is thought to be associated with sudden death. Such a practice, no doubt, has been responsible for some of the dogmatic statements that no such entity exists. For instance, the Status Lymphaticus Investigation Committee of the British Medical Association concluded that there was no evidence that so-called "status thymicolymphaticus" had any existence as a pathologic entity.[3]

*This term is used instead of status thymicolymphaticus because, as Marine states, the latter overemphasizes the importance of the thymus in this syndrome.

Greenwood and Woods[32] even go so far as to state that "status lymphaticus is an example of the growth of medical mythology." Marine,[2] while accepting the view of many that there is no known disease entity in which the thymus plays a central or causal rôle, believes that there is a syndrome characterized anatomically by lymphoid hyperplasia in which the essential factor is lowered resistance or increased susceptibility. Undoubtedly the medical profession has been too willing to attribute unexplained sudden death in infants and children to thymic death, but, nevertheless it does occur in individuals, not always of the overnourished type, in whom nothing but hyperplasia of the lymphoid structures is found at autopsy. While death in these cases may be due to causes undeterminable by the methods used at present, it would seem that the lymphoid changes, which have been found so consistently through the years, must be related in some manner to the condition. All cannot agree with Boyd[5]: "The anatomic picture called status thymicolymphaticus is the normal state of the healthy person." Certainly it seems that the burden of proof regarding the nonexistence of this controversial syndrome should rest with the doubters, who must develop a technic capable of determining the cause of these sudden deaths. Some attempts have been made along such lines. Death in these cases has been likened sometimes to death from anaphylactic shock.[33] Recently Waldbott[34] has reviewed this subject again. Admitting that a large number of so-called thymic deaths are misdiagnosed he was able to isolate 34 out of 102 cases which he thought conformed to the picture. In all of these there were uniform changes which suggested that death was due to primary anaphylactic edema of the lungs and asphyxia. Furthermore, in a group of 30 living infants diagnosed clinically as thymic children 24 were found to be afflicted with allergic disease. It has been shown also that thymolymphatic hyperplasia is associated with thyroid hypersecretion[35] and suprarenal deficiency.[36] Recently Szabados[15] has tried to include all of these associated factors in the hypothesis that the primary cause is tissue asphyxia. Such reports, rather than offering a complicating obstacle to the study of the problem, seem to lend support to the existence of a syndrome of this nature. They also serve to indicate that the possible solution may be reached ultimately by probing more deeply into those intriguing, but little understood, and more or less closely allied subjects, the endocrines, allergy, and the autonomic nervous system. Whether or not the ephemeral condition loosely termed "status lymphaticus" and "lymphatic constitution" by older writers will finally be proved to exist cannot be foretold for so far the case for or against has not been closed. While at the present time the condition too often is made the dumping ground for unexplained sudden deaths, one would hesitate to say positively that it does not occur. It is very likely that in the future the term will be replaced by one more descriptive of the underlying cause.

OTHER DISEASES OF THE THYMUS

Dubois' Abscesses.—This rarity, which has not been diagnosed clinically, consists of small, sharply defined cavities filled with pus from which spirochetes usually can be obtained. The cysts develop primarily by degeneration of lymphocytes and Hassall's corpuscles followed by hypertrophy of the reticular epithelium and fusion of the degenerated corpuscles.[37] It has been generally conceded that the origin of these

cysts or abscesses is syphilitic. Recently Benjamin[38] reported a case with lesions indistinguishable from typical Dubois' abscesses in which there was no evidence of syphilitic origin. Maternal thyrotoxicosis was suggested as a possible etiologic agent.

Thymic Apoplexy.—Owing to the rich vascular supply of the thymus, congestion and hemorrhage is likely to occur whenever there is circulatory stasis. Friedleben applied the term "thymic apoplexy" to the extensive hemorrhages found in the newborn. He assumed that the trauma of difficult labors was the chief etiologic factor, but others have attributed it to syphilis, circulatory stasis, and the hemorrhagic tendency of the newborn.[5] It usually occurs in very young infants and often results in death. Such cases probably are not as rare as the literature would lead one to think.

THYROID GLAND

Greater interest in the effect of thyroid disease upon the gravid state has led to an increasing number of studies about the fetal and neonatal thyroid. At birth the normal gland is relatively large, and generally in goitrous districts its size is increased. Histologically the thyroid of the fetus and newborn differs little from the adult except for the size of the follicles.[39] During pregnancy activity of the maternal gland is increased.[40, 41] The gland is active during the latter months of fetal life,[42] but how adequate the secretory function is has not been ascertained definitely. The presence of hyperactivity of the maternal gland during pregnancy and the lack of symptoms for some weeks after birth in cases of aplasia of the neonatal thyroid indicate that some secretion is supplied the fetus during intrauterine life and that the child is born with a supply.[43] While there is some dispute over the iodine content of the fetal thyroid, the amount, if present, is very small.

Prenatal Iodine Deficiency.—Iodine deficiency in the maternal diet increases the incidence of congenital goiter.[44, 45, 46, 47] Therefore, in goitrous districts iodine should be administered antenatally for the sake of the fetus as well as the mother. Both maternal goiter and hypothyroidism[48] are considered responsible for congenital goiter.

Thyroid Hyposecretion.—Hypoplasia or aplasia of the thyroid is uncommon. In the United States only the sporadic form is seen, but in certain localities in Europe in which goiter is endemic it may be endemic also. The terms "cretinism" and "congenital myxedema" are used to signify the symptom-complex of hypothyroidism. No doubt there are many cases of mild hypothyroidism that pass unrecognized. Although cretinism is a congenital disease, it is noticed rarely at birth[49] probably because the infant is born with a sufficient supply of thyroxin to last him a while. Furthermore, it is likely that some of the secretion may be supplied by the breast milk, for symptoms appear earlier in artificially fed infants.[50] The principle symptoms consist of thickness of the tongue, apathy, failure to gain, delayed growth with retardation of the appearance of the ossification centers, hoarseness, and a peculiar but characteristic infiltration of the subcutaneous tissue. These symptoms are difficult to recognize in the neonatal period and if observed may be confused with mongolism which also manifests signs somewhat suggestive of thyroid insufficiency. When such findings are detected at birth, the condition is more likely to be mongolism. The need for early diagnosis and treatment has been emphasized by Talbot,[11] who states that the

cretinism is recognizable in the third month of life. The longer treatment is delayed the less the chances to obtain complete normality. Even so, treatment is more successful from the physical than the mental standpoint. Thyroxin is the therapeutic agent of choice because it is very potent and its dosage can be accurately controlled. In localities where cretinism is endemic the use of 0.1 gram of iodine during the last five months of pregnancy is said to be a prophylactic.[51] However, there is no evidence so far to show that sporadic cretinism is due to iodine deficiency.

Thyroid Hypersecretion.—This type of thyroid disease is practically unheard of in the newborn. White[52] is the only one who has reported exophthalmic goiter at this age. The infant died on the second day. Klaus,[53] Helmholz,[54] and Elliott[55] have described cases in infants under one year of age. Partial thyroidectomy was performed upon Elliott's patient at the age of two and one-half years.

Thyroid Enlargements.—Transitory swelling of the thyroid is observed occasionally. It is present at birth or appears shortly afterward as a moderately sized, soft, and rather uniform enlargement which disappears in a few days. It is thought to be due to hyperemia from pressure upon the gland and adjacent structures during birth. The fact that it is more common in face presentations indicates this etiology. Spontaneous massive hemorrhage sufficient to cause death has been observed by Mutel and Morin.[56]

Congenital Goiter.—It is generally agreed that the most important factor in the production of congenial goiter is deficiency of iodine in the diet. For that reason the incidence varies in different localities. Regions where there is iodine deficiency in the water are known as goitrous belts. Mothers of infants with congenital goiters usually have similar enlargements.[57, 58] The gland is not always uniformly enlarged in the fetus, and usually the goiters are small although occasionally they may reach tremendous size,[59] sufficient to obstruct labor.[60] Most commonly the pathology consists of hyperplasia of the parenchyma, and associated usually with the tissue increase is a varying degree of transient hyperemia. When the goiter is large, the adjacent tissues may be pressed upon, and the trachea may be flattened. Pressure sufficient to prevent the normal development of the cartilaginous rings has been noted.[61] Enlargement of the thymus is a rather commonly associated finding. Symptoms are absent in most instances, but, if the goiter is large, signs of obstruction and cyanosis may be observed, and death from asphyxia may occur. Owing to concomitant transitory swelling, the obstructive symptoms are worst a few hours after birth, and if the infant does not die in thirty-six hours, his chances of living are good. After this period the hyperemic swelling disappears rapidly. Gradually the goiter subsides, and especially is this true when iodine therapy is instituted. Sometimes, however, it persists[57] or continues to grow until operative intervention is required.[62] As a rule, toxicosis is not observed in congenital goiter.

Prophylactic treatment is most important and is accomplished by administration of iodine before and during pregnancy in some of the commonly used forms.[39, 40, 45, 47] Active treatment consists of relief of transitory swelling by cold applications and extension of the head. For respiratory embarrassment the air passages should be kept clear of

discharges and oxygen supplied. When obstructive symptoms threaten the life of the infant, tracheal catheterization or possibly intubation should be resorted to. Tracheotomy at this age is contraindicated. Section of the isthmus is to be preferred to thyroidectomy, which carries a high mortality rate. In the majority of cases, however, such measures are unnecessary because symptoms are very mild or absent. Reduction of the goiter may be brought about by administration of iodine to the infant. In some instances the response is prompt.[63] Sweet[59] was able to reduce a large goiter in a breast-fed infant by administering iodized calcium lactate to the mother.

The incidence of congenital goiter should be materially decreased in this country owing to the rather general use of iodized salt in goitrous localities.

SUPRARENAL GLANDS

These structures are relatively large at birth, but in the next two weeks cortical involution causes considerable reduction in size. Since traces of epinephrine may be found in the glands during the late fetal life and at birth, there is reason to believe that there is some activity present at these times.[42] However, there have been so few physiologic studies in early life that little can be said about the action of the suprarenals in the fetus and newborn. Even at a later period most of the information we possess is largely of an academic nature, more interesting to the physiologist than to the practitioner. However, as will be seen later, considerable clinical interest should be manifest about the probable rôle of the suprarenal cortex in heat production[64, 65] and the regulation of respiration.[66, 67] Cortical insufficiency is said to be associated with thymic enlargement in animals.[2, 36, 68] In the human the rather common observation that the suprarenals of infants with the so-called syndrome status lymphaticus are smaller than normal seems to bear this out. Furthermore, the reports that the blood sugar level in cases of thymic enlargement[22] and status lymphaticus[69] is low serve to support this view. It seems entirely possible that the cortical involution of the suprarenals, which begins in the second week of extrauterine life, may be a factor in the production of hyperplasia of the thymus and lymph glands in early infancy.

Cortical hyperplasia associated with virilism has been reported frequently and is due to benign or malignant tumors of the suprarenal cortex.[70] Many of these growths while not recognized early are actually of congenital origin.[71, 72] The signs of virilism caused by suprarenal tumors have been observed as early as eighteen weeks.[73] Prompt diagnosis is essential because metastasis occurs very early. A few successful extirpations have been performed in young infants.[74, 75]

Hemorrhage.—Suprarenal hemorrhage is the most common affection of these glands in the neonatal period. In fact most of such hemorrhages are found at this particular time of life. Owing to the vascularity of the glands, hyperemia of the cortical and capsular portions is a frequent observation at autopsy, and histologically small hemorrhages may be detected in nearly all newborns. The largest number of cases has consisted of massive hemorrhages found in stillborn or in infants dying shortly after birth,[76] but probably a minor form occurs in many others

that survive. As a rule, suprarenal hemorrhage is diagnosed only at autopsy, but in a few instances it has been recognized in the living infant.[77, 78, 79, 80]

The bleeding is considered by many to be due to trauma and to an unusual susceptibility of the suprarenals to hemorrhage. Trauma may come from difficult labors, the pressure occurring during normal labors, asphyxia, and the use of artificial respiration for resuscitation. The hemorrhagic tendency of the newborn has been considered a possible cause. The peculiar susceptibility of the suprarenals to hemorrhage has been explained by the rapid involution that occurs in late fetal life or immediately after birth. Disappearance of many of the cortical cells leaves the thin-walled capillaries unsupported and more subject to rupture. Thus the anatomic structure of the glands together with the severity of trauma determines the occurrence and degree of hemorrhage. The hemorrhage may not extend beyond the capsule, leaving the gland enlarged but firm. Later the gland may become cystic. When the capsule ruptures, the blood usually extravasates into the retroperitoneal tissue although occasionally it may empty into the peritoneal cavity. In old hemorrhages of minor severity hemosiderin and calcium deposits may be found.

The symptoms vary with the extent of bleeding and the amount of damage to the parenchyma of the gland. The chief symptoms are shock, rapid respiration, and hyperpyrexia in the presence of normal lungs and palpation of a progressively increasing mass in either flank. In a few cases fever has not been observed.[80] Other symptoms consist of convulsions, skin petechiae, and progressive anemia. Arnold[80] considers a rapid fall of the hemoglobin index of diagnostic importance. A very low blood sugar level suggests impairment of the suprarenals, probably the medullary portion.[81] If the hemorrhage is unilateral, signs of acute insufficiency may or may not be present.

While spontaneous recovery probably occurs in the mild cases, death is the usual outcome when hemorrhage is massive. Treatment consists of immediate transfusion, and in cases in which the signs of severe concealed hemorrhage are outstanding possibly surgical intervention may be justified. Continued administration of cortical hormone (cortin) is necessary to relieve the symptoms of acute cortical insufficiency.

The syndrome of suprarenal hemorrhage, which is characterized apparently by the cardinal symptoms of shock, rapid respirations, and hyperpyrexia, may be the cause of some of the unexplained sudden deaths occurring shortly after birth. Closer attention to the symptoms manifest by newborn infants might result in more frequent diagnoses of this condition and with prompt treatment lives might be saved. Further study of this question may reveal valuable information about milder hemorrhages which now escape detection.

PANCREAS

The literature contains few references to the pancreas of the newborn. Histologically the gland is more vascular than in later life; the alveoli are fewer; and the number of islands of Langerhans is relatively greater. The gland at birth is said to contain all the ferments found in the adult but in smaller amounts. Minute subcapsular hemorrhages are frequent at this time, and fatal rupture has been reported. Fibrosis

due to syphilis is observed occasionally. The gland is enlarged and firm, and microscopically there is an excessive perivascular connective tissue overgrowth, and in some instances diabetic symptoms appear.[82] The nonspecific form results from congenital atresia of the pancreatic duct,[83] which often is associated with atresia of the bile ducts.[84] Pancreatic fibrosis produces congenital steatorrhea with or without infantilism.[85, 86] In this disease the stools are soft, large, pale, and greasy. Fat droplets are visible grossly and microscopically many fat droplets, but no fatty acid crystals are seen. Owing to the deranged digestive function, malnutrition and dwarfism occur. Abscesses, cysts, and neoplasms of the pancreas of the newborn are very rare.

The clinician's interest in this gland chiefly concerns its internal secretion. Metabolic studies[87] have shown that the fetal carbohydrate supply at birth is not great so that increased consumption of it by chilling and muscular effort should be prevented wherever possible. Theoretically, at least, acidosis may be prevented by administration of carbohydrate in some form until the breast milk supply is adequate. Diabetes is uncommon in infancy and especially rare in the newborn. The antithesis of diabetes, hyperinsulinism, has been reported in infants of diabetic mothers.[88, 89, 90] The hypoglycemic manifestations consist of weakness, irritability, and convulsions.

While usually there is little tendency upon the part of the fetal pancreas to compensate for the deficiency of insulin in the diabetic mother, such reports indicate that it may occur at times. Occasionally it is necessary to decrease the dosage of insulin in the latter months of pregnancy because of this.[91] We have observed such a case.[1] At autopsy the glands of infants dying of hyperinsulinism from this cause showed hypertrophy and hyperplasia of the gland and a marked increase in the number of islets.[92] Hypoglycemia, however, may occur in infants whose mothers are not diabetic.[22] The blood sugar level may be 50 mg. per hundred cubic centimeters or less. However, it must be remembered that the blood sugar of the apparently normal newborn infant frequently is as low as that mentioned above. Maternal diabetes seems to be responsible, in some instances at least, for excessive fetal growth. Skipper[93] reported that 18 per cent of the infants of diabetic mothers weighed over 10 pounds. Bix,[94] who found that 8 per cent weighed 9 pounds and nearly 11 per cent over 10 pounds, emphasizes a point of clinical interest, namely, that the birth of an oversize baby should lead the physician to suspect potential diabetes in the mother. The overgrowth is attributed to an elevation of the blood sugar level.

COLIC (GASTROENTEROSPASM, HYPERTONIA)

It is with the hope of stimulating further research that the reviewer has the temerity to broach upon the subject of colic, a syndrome about which so much is heard but so little written. In spite of the fact that some pediatrists emphatically deny its existence, others whose practices are not limited to the hospital continue to recognize it as a clinical entity. Certainly the laity does and, furthermore, not only expects the practitioner to do likewise but also to treat it. Recognizing full well that the term "colic" may be a misnomer, I will not attempt to quibble about a name. There does exist in the first few weeks or more of life a syndrome consisting of hyperirritability, fussiness, increase of skeletal muscle tone, fist-sucking, belching, regurgitation or vomiting, and at-

tacks of acute distress that appear to be located in the abdomen. In order to determine more definitely the possible cause of some of these symptoms, they may be divided into two general groups: local and constitutional.

Local Causes.—This group may be subdivided into those the cause of which appears to lie within or without the gastrointestinal tract, that is, enteral or parenteral causes.

In the enteral group the acute attacks seem to be centered in the gastrointestinal tract and are associated with tympanites, for, when it is relieved, the distress subsides. The stools are unhomogeneous and of greenish color, and contain many curds as well as a varying amount of mucus. To this condition Finkelstein applied the term "dyspepsia." In a goodly number of infants so affected the cause seems to be a quantitative one of overfeeding. This is substantiated by the fact that they make excessive gains and restriction of the amount of food consumed produces prompt relief. At other times distress is not allayed in this manner. Because of the large amount of flatus the feeding error has been considered a qualitative one due either to high fat or carbohydrate consumption or to a decreased tolerance for these foodstuffs. To remedy this situation, reduction of the concentration of these substances or dilution of them by increasing the protein content of the food has been advocated.[95]

In the parenteral group the gastrointestinal symptoms may not be as prominent, and frequently a thorough examination will locate disease elsewhere in the body. Nasopharyngitis, otitis media, and pyelitis are the most common, but other disturbances of a more obscure nature may finally be detected. Platou[96] mentions the possibility that herniation through peritoneal and fascial defects may cause these symptoms and reports such a case in which finally strangulation occurred.

Constitutional Causes.—Sometimes the symptoms of so-called colic do not respond to the measures mentioned above, and there is no evidence of disease elsewhere. One is then confronted with the possibility that crying has become a habit or to put it bluntly the infant is "spoiled." It is difficult and often impossible to determine where so-called colic ends and spoiling begins. No doubt, in most instances both may exist together. Environment probably plays an important part in the production of these symptoms, for the infants usually come from homes of so-called nervous families in which the parents and other relatives are so unduly apprehensive about the baby that there is a profusion of excitement when he cries. Usually these infants are firstborn. The temperament and emotional instability of the parents of such children often makes it possible to predict prenatally that the baby will be of this type. Observations of this nature may be responsible for the lay concept of prenatal influence. Even heredity has been incriminated, and some have considered this state a constitutional one, the so-called neuropathic infant.

Reports from time to time have directed attention to other factors which may play a rôle in the production of some of these symptoms, especially in infants who do not conform exactly to the picture described above. They are fussy and do not gain well, and their stools frequently are constipated rather than loose. Several terms have been used to describe this state, which in some ways simulates the vagotonia of Ep-

penger and Hess. Thus Haas[97] called it hypertonia and White[98] vagogenic gastroenterospasm. Varying degrees of cardiospasm and pylorospasm may be associated.[98, 99, 100, 101] All of these terms relate essentially to the same condition, one in which there is thought to be an autonomic imbalance, that is, either hyperactivity of the parasympathetics (craniosacral outflow) or hypoactivity of the sympathetics (thoracicolumbar outflow).[98] The basis for reasoning this way is the response to atropine, which, when given in doses sufficient to cause definite flushing of the face, relieves the symptoms. Other measures are advised. The infants should be cared for by other persons than the parents in order to avoid spoiling, air swallowing should be corrected, and dietary measures instituted. In this condition as with pylorospasm, in which atropine is used, thick cereal feedings are of value. Some have found phenobarbital exceptionally valuable.[102] In our hands this drug has been most useful in relieving both the actual distress and the spoiling which is invariably present to some degree.

There seems to be some relationship between vagotonia and allergy.[103] In both there seems to be a basic derangement of the autonomic nervous system in which possibly endocrine dysfunction, suprarenal deficiency, and a disturbed calcium-potassium balance are involved. Similarly the same drugs, atropine, epinephrine, and calcium, are effective in allergic conditions as well as in vagotonia. Others[104, 105, 106] have emphasized the allergic factor in this syndrome, particularly in respect to the transmission of food proteins through the breast milk.[104, 107] Furthermore, the fact that eczema, which has been added to the list of allergic diseases, frequently is associated with gastroenterospasm, tends to support the idea that allergy may play a part in both.[103, 106] Mayerhofer proposed the name, "the allergic syndrome of the newborn infant," for some of these manifestations at this age, among which he included most of the beginning disturbances of the gastrointestinal tract and pylorospasm.[108]

The symptoms of gastroenterospasm have been linked with tetany, too.[109] A few reports have appeared about tetany in the newborn[110, 111, 112, 113] in which to the usual manifestations of tetany (carpopedalspasm, laryngospasm, positive Chvostek sign, and convulsions) are added nervous irritability, hypertonia, and in some instances such gastrointestinal symptoms as projectile vomiting and dysphagia. Even some have included contraction of smooth muscle (smooth muscle tetany) in the picture,[109] but this is open to question. The absence of electrical tests and blood calcium determinations in some of the case reports as well as the fact that the Chvostek sign may be found in normal infants[114] has led to some dispute about these cases. Others have wondered whether the symptoms as described are primarily due to tetany or to vomiting initiated by intracranial pressure, hypertonia, and other disturbances.[1] Some years ago Ylppö described a spasmophilia from cerebral injury in premature infants, and von Reuss observed definite symptoms of tetany in an infant who died of subdural hemorrhage.[115] In some ways the symptoms described under tetany of the newborn are similar to those of hypertonia, vagotonia, or gastroenterospasm, which some of the same writers formerly thought to be due to an allergic factor.

However, it is not the purpose here to do more than outline the difficulties involved in this controversial subject. Associated with this loosely grouped aggregation of symptoms known as colic are a diversity of more or less closely related factors, autonomic imbalance, allergy, and possibly the endocrines.

Regardless of the cause or of the numerous other terms used to describe it, colic is colic to the layman and demands correction. Treatment consists of determining the cause wherever possible. If local parenteral causes are excluded and the symptoms point to a feeding error, curtailment of food or restriction of the excess of some of the foodstuffs is indicated. When this does not seem to be the case and hypertonia exists, atropine sulphate in physiologic doses or phenobarbital probably will reduce spasm, correct the psychic factor, allay hyperirritability, and afford the infant as well as the attendants their much needed sleep. When there is evidence of allergic manifestations, the cause of these should be sought and removed, and in case there is evidence of calcium deficiency this mineral should be administered in some quickly available form.

REFERENCES

1. Bonar, B. E.: Problems of the Newborn, J. PEDIAT. **5**: 216, 1934.
2. Marine, D.: Status Lymphaticus, Arch. Path. **5**: 661, 1928.
3. Young, M., and Turnbull, H. M.: An Analysis of the Data Collected by the Status Lymphaticus Investigation Committee, J. Path. & Bact. **34**: 213, 1931.
4. Capper, A., and Schless, R. A.: The Thymus Gland and Thymic Symptoms, J. PEDIAT. **4**: 573, 1934.
5. Boyd, E.: The Growth of the Thymus, Am. J. Dis. Child. **33**: 867, 1927.
   Idem: The Weight of the Thymus in Health and in Disease, Ibid. **43**: 1162, 1932.
6. Park, E. A., and McClure, R. D.: Results of the Thymus Extirpation in the Dog, Am. J. Dis. Child. **18**: 317, 1919.
7. Rowntree, L. G., et al.: The Biologic Effects of the Thymus Extract (Hanson's), J. A. M. A. **19**: 1425, 1934.
8. Wahl, H. R., and Walthall, D.: Thymus Apoplexy, Am. J. Dis. Child. **24**: 27, 1922.
9. Foot, N. C., and Harrington, H.: Malignant Epithelial Thymoma, Am. J. Dis. Child. **26**: 164, 1923.
10. Noback, G. J.: A Contribution to the Topographic Anatomy of the Thymus Gland, etc. Am. J. Dis. Child. **22**: 120, 1921.
11. Talbot, F.: Disorders of Internal Secretion in Children, J. PEDIAT. **1**: 766, 1932.
12. Hart, C.: Pathology of the Thymus, Arch. f. path. Anat. **214**: 1, 1913.
13. Pancoast, H. K., and Pendergrass, E. D.: The Modern Roentgenologic Aspect of the Thymus in Infancy, Am. J. Dis. Child. **42**: 192, 1931.
14. Pancoast, H. K.: Roentgenology of the Thymus in Infancy, Am. J. Dis. Child. **40**: 452, 1930.
15. Szabados, M.: Status Thymicolymphaticus, J. PEDIAT. **4**: 798, 1934.
16. Blackfan, K. D., and Little, K.: A Clinical and Radiographic Study of the Thymus in Infants, Am. J. Dis. Child. **22**: 459, 1921.
17. De Buys, L. R., and Samuel, E. C.: A Study of the Shadows in the Thorax of the Newly-Born, Am. J. Dis. Child. **24**: 397, 1922.
18. Liss, I. E.: The Thymic Shadow in Infants, Am. J. Dis. Child. **24**: 192, 1922.
19. Greenthal, R. M.: The Incidence of Thymic Enlargement in Infants and Children, Am. J. Dis. Child. **24**: 433, 1932.
20. Gerstenberger, H. J.: The Factor of the Position of the Diaphragm in Roentgen-Ray Diagnoses of Enlarged Thymus, Am. J. Dis. Child. **21**: 534, 1921.
21. Hasley, C. K., and De Tomasi, R. Q.: Cin-ex Camera Studies of Thymus in Infants and Children, J. Michigan M. Soc. **29**: 25, 1930.
22. Aldrich, C. A., and Munns, G. F.: Hypoglycemia in Infants, Am. J. Dis. Child. **42**: 694, 1931.

23. Wasson, W. W.: Thymic Stridor, J. A. M. A. **89**: 1025, 1927.
24. Mitchell, A. G.: Panel Discussion on Endocrine Glands, J. PEDIAT. **6**: 860, 1935.
25. Waldbott, G. L., and Anthony, G. E.: So-called Thymic Hyperplasia, Am. J. Dis. Child. **47**: 34, 1934.
26. Kennedy, R. L., and New, G. B.: Chronic Stridor in Childhood, J. A. M. A. **96**: 1286, 1931.
27. New, G. B.: Congenital Obstruction of the Larynx and Pharynx, J. A. M. A. **81**: 363, 1923.
28. New, G. B., and Clark, C. M.: Angioma of the Larynx, Ann. Otol., Rhin. & Laryng. **28**: 1025, 1919.
29. Tucker, G.: The Infant Larynx, J. A. M. A. **99**: 1899, 1932.
30. New, G. B., and Clark, C. M.: Angiomas of the Larynx, Ann. Otol., Rhin. & Laryng. **30**: 631, 1921.
31. Barnes, J. M.: Late Effects of Treatment of Thymus, Am. J. Roentgenol. **29**: 220, 1929.
32. Greenwood, M., and Woods, H. M.: Status Thymicolymphaticus Considered in the Light of Recent Work on the Thymus, J. Hyg. **26**: 305, 1927.
33. Symmers, D.: Status Lymphaticus, Am. J. M. Sc. **156**: 40, 1918.
34. Waldbott, G. L.: So-Called Thymic Death, Am. J. Dis. Child. **47**: 41, 1934.
35. Hoskins, G.: Congenital Thyroidism, Am. J. Physiol. **26**: 426, 1910.
36. Marine, D., et al.: Influence of Thyroidectomy, Gonadectomy, Splenectomy, on Thymus Gland of Rabbits, J. Exper. Med. **40**: 429, 1924.
37. Oliver, J.: Syphilitic Disease of the Thymus in Infants and Mode of Origin in Dubois' Abscesses, Am. J. Dis. Child. **13**: 158, 1917.
38. Benjamin, E. L.: Dubois' Sequestra of the Thymus Gland of Nonsyphilitic Origin, Am. J. Dis. Child. **39**: 586, 1930.
39. Abbott, A. C., and Ball, R. P.: Pathology of the Thyroid Gland of the Human Fetus and the New Born Infant, Canad. M. A. J. **24**: 347, 1931.
40. Soule, S. D.: A Study of the Thyroid Activity in Normal Pregnancy, Am. J. Obst. & Gynec. **23**: 165, 1932.
41. Fleischer, A. J.: Thyrotoxicosis Complicated by Pregnancy, Am. J. Obst. & Gynec. **22**: 273, 1931.
42. Kraus, E. J.: Aplasia of the Thyroid in Relation to Endocrine Function During Fetal Life, Beitr. z. path. Anat. u. z. allg. Path. **82**: 291, 1929.
43. Schiff, E.: Thyroid Gland, Monatschr. f. Kinderh. **27**: 359, 1924.
44. Hill, A. L.: Congenital Goiter, Arch. Pediat. **50**: 424, 1933.
45. Marine, D.: Thyroid Gland in Gynecology and Obstetrics, Surg. Gynec. Obst. **25**: 272, 1917.
46. Davis, C. H.: The Prophylactic Treatment of Thyroid Dysfunction and the Importance of Basal Metabolism Studies in Obstetrics and Gynecology, Am. J. Obst. & Gynec. **24**: 607, 1932.
47. Wallace, J. T.: Thyrotoxicosis in Its Relation to Pregnancy, Am. J. Obst. & Gynec. **26**: 77, 1933.
48. Litzenberg, J. C., and Carey, J. B.: Relation of the Basal Metabolism to Gestation, Am. J. Obst. & Gynec. **17**: 550, 1929.
49. Lohnes, H.: A Case of Congenital Myxedema in a Breast-Fed Infant With Unusually Early Manifestations of Symptoms, Bull. Buffalo Gen. Hosp. **3**: 17, 1925.
50. Lukacs, J.: Is the Thyroid Hormone Secreted in the Mother's Milk? Arch. f. Kinderh. **91**: 9, 1930.
51. Hanziker, H., and Eggenberger, H.: Quoted by Mellanby, E., in "Nutrition and Child-Bearing," Lancet **2**: 113, 1933.
52. White, C.: A Fetus With Congenital Hereditary Graves' Disease, Proc. Roy. Soc. Med. (Obst. Sect.) **5**: 247, 1912.
53. Klaus, O.: Basedow's Disease in a Nine-Month-Old Child, Prag. med. Wchnschr. **39**: 315, 1914.
54. Helmholz, H. F.: Exophthalmic Goiter in Childhood, J. A. M. A. **87**: 157, 1926.
55. Elliott, P. C.: Exophthalmic Goiter Before One Year of Age, J. PEDIAT. **6**: 204, 1935.
56. Mutel and Morin: Death at Birth From Hemorrhage Into the Thyroid Gland, Ann. de méd. lég. **8**: 157, 1928 (Abst. Am. J. Dis. Child. **39**: 850, 1930). 1930).
57. Skinner, H. H.: Congenital Goiter, J. A. M. A. **82**: 1190, 1924.
58. Porter, W. B., and Vanderlehr, R. A.: Congenital Goiter, Am. J. Dis. Child. **22**: 477, 1921.

59. Sweet, P. W.: Goiter in a New-Born Infant, Northwest Med. 27: 183, 1928.
60. McLannahan, J. G.: Fetal Goiter, Brit. M. J. 1: 712, 1913.
61. Skinner, G. A.: Results in Congenital Goiter, M. J. & Rec. 127: 381, 1928.
62. Williamson, G. R.: Congenital Goiter, J. PEDIAT. 4: 458, 1933.
63. Hamburger, F.: The Treatment of Goiter in Infants, München. med. Wchnschr. 71: 1815, 1924.
64. Marine, D., and Bauman, E. J.: Influence of Glands With Internal Secretions on Respiratory Exchange, Am. J. Physiol. 59: 353, 1922.
65. Cramer, W.: Fever, Infections, and Thyroid-Adrenal Apparatus, Brit. J. Exper. Path. 7: 95, 1926.
66. Bornstein, A., and Holm, K.: The Respiration After Suprarenal Extirpation, Ztschr. f. d. ges. exper. Med. 37: 1, 1923.
67. Kühl, G.: Researches on the Action of the Suprarenal Hormone, Arch. f. d. ges. Physiol. 215: 277, 1927.
68. Jaffé, H. L.: The Influence of the Suprarenal Gland on the Thymus: Parts I and II, J. Exper. Med. 40: 325, 619, 1924.
69. McLean, A. B., and Sullivan, R. C.: Blood Sugar in Status Thymicolymphaticus, Am. J. M. Sc. 171: 659, 1926.
70. Talbot, F.: Disorders of the Internal Gland Secretion in Children, J. PEDIAT. 1: 766, 1932.
71. Cecil, H. L.: Hypertension, Obesity, Virilism, and Pseudohermaphroditism, J. A. M. A. 100: 463, 1932.
72. Van der Bogert, F.: Virilism and Hypertension in Infancy Associated With Adrenal Tumor, J. PEDIAT. 4: 629, 1933.
73. Lightwood, R.: Tumor of the Suprarenal in an Infant of 18 Weeks, Arch. Dis. Child. 7: 37, 1932.
74. Lehman, E. P.: Adrenal Neuroblastoma in Infancy, Ann. Surg. 95: 473, 1932.
75. Collett, A.: Genito-Suprarenal Syndrome in a Girl One and a Half Years Old With Successful Operation, Am. J. Dis. Child. 27: 204, 1924.
76. Browne, F. J.: Neonatal Death, Brit. M. J. 2: 590, 1922.
77. Corcoran, W. J., and Strauss, A. A.: Suprarenal Hemorrhage in the Newborn, J. A. M. A. 82: 626, 1924.
78. Goldzieher, M. A., and Greenwald: Hemorrhage of the Suprarenals in a New-born Infant, Am. J. Dis. Child. 36: 324, 1928.
79. Rosenblum, J.: Suprarenal Hemorrhage in the New-born Infant, Am. J. Dis. Child. 43: 662, 1932.
80. Arnold, D. P.: Massive Suprarenal Hemorrhage in the Newborn Infant, Am. J. Dis. Child. 40: 1053, 1930.
81. Greenwald, H. M., and Pennell, S.: The Carbohydrate Metabolism of the Normal Newborn Infant, Am. J. Dis. Child. 39: 281, 1930.
82. Menninger, W. C.: Congenital Syphilis of the Pancreas, Am. J. Syph. 13: 527, 1929.
83. Kornblith, B. A., and Otani, S.: Meconium Ileus With Congenital Stenosis of the Main Pancreatic Duct, Am. J. Path. 5: 249, 1929.
84. Higgins, T. T.: Subacute Pancreatitis Occurring in Association With Acute Intestinal Obstruction in New-born Infant, Brit. J. Surg. 11: 592, 1924.
85. Clarke, C., and Hadfield, G.: Congenital Pancreatic Disease With Infantilism, Quart. J. Med. 17: 358, 1924.
86. Harper, M. H.: Two Cases of Congenital Pancreatic Steatorrhea With Infantilism, M. J. Australia 2: 663, 1930.
87. Benedict, F. G., and Talbot, F. B.: The Physiology of the New-born Infant, Publication 233, Washington, D. C., 1915, Carnegie Institute of Washington.
88. Wiener, H. J.: Diabetes Mellitus and Pregnancy, Am. J. Obst. & Gynec. 7: 710, 1924.
89. Gray, S. H., and Feemster, L. C.: Compensatory Hypertrophy and Hyperplasia of the Islands of Langerhans in the Pancreas of a Child Born of a Diabetic Mother, Arch. Path. & Lab. Med. 1: 348, 1926.
90. Nothman, M., and Hermstein, A.: Diabetes and Pregnancy, Arch. f. Gynäk. 150: 287, 1932.
91. Parson, P., et al.: Pregnancy and Diabetes, M. Clin. North America 10: 679, 1926.
92. Heiberg, K. A.: The Pancreatic Islets of the New-born Infant of a Diabetic Mother, Virchows Arch. f. path. Anat. 287: 629, 1933.
93. Skipper, E.: Diabetes Mellitus and Pregnancy, Quart. J. Med. 2: 353, 1933.

94. Bix, H.: The Relation Between Maternal Diabetes and Infant Birth Weight, Med. Klin. 29: 50, 1933.
95. Grulee, C. G.: A Treatment for Colic in the Breast-Fed Infant, J. A. M. A. 75: 1701, 1920.
96. Platou, E. S.: Possible Anatomic Basis for Certain So-Called Cases of Colic in Infants, Arch. Path. 44: 458, 1927.
97. Haas, S. B.: Hypertonic Infant, Am. J. Dis. Child. 15: 323, 1918.
98. White, P. J.: Gastroenterospasm as a Manifestation of Autonomic Imbalance in Early Infancy, Am. J. Dis. Child. 26: 91, 1923.
99. Lippman, H. S.: Restlessness in Infants, J. A. M. A. 91: 1848, 1928.
100. Birnberg, T. L.: Cardiospasm in the New-born Infant, Am. J. Dis. Child. 38: 1183, 1929.
101. Segar, L. H., and Stoeffler, W.: Cardiospasm in the New-born Infant, Am. J. Dis. Child. 39: 354, 1930.
102. Barnett, E. J.: The Use of Phenobarbital in Infant Feeding, Arch. Pediat. 47: 452, 1930.
103. Klauder, J. V.: Clinical Aspects of Allergy, Arch. Dermat. & Syph. 19: 198, 1929.
104. O'Keef, E. S.: The Relation of Food and Infantile Eczema, Boston M. & S. J. 182: 569, 1920.
105. Shannon, W. R.: Neuropathic Manifestations in Infants and Children as a Result of Anaphylactic Reactions to Foods Contained in Their Dietary, Am. J. Dis. Child. 24: 89, 1923.
106. White, P. J.: The Relation Between Colic and Eczema in Early Infancy, Am. J. Dis. Child. 38: 935, 1929.
107. Shannon, W. R.: Demonstration of Food Proteins in Human Breast Milk by Anaphylactic Experiments on Guinea Pigs, Am. J. Dis. Child. 22: 223, 1921.
108. Mayerhofer, E.: The Allergic Syndrome of the Newborn Infant, Ztschr. f. Kinderh. 49: 572, 1930.
109. Lemaire, H., and Oliver: Habitual Vomiting and Tetany, Nourrisson 10: 305, 1922.
110. Shannon, W. R.: Generalized Edema in Association With Tetany of the New-Born, Arch. Pediat. 46: 549, 1929; Tetany, Generalized Edema and Cerebral Compression in the New-born, Ibid. 48: 153, 1931; and Cerebral Injury in the New-born, Am. J. Dis. Child. 48: 517, 1934.
111. Bass, M. H., and Karelitz, S.: Tetany Accompanied by Hyperpyrexia and Vomiting in the First Days of Life, J. A. M. A. 97: 1372, 1931.
112. McGavaran, E. G.: Spasmophilic Tetany in the Newborn, J. A. M. A. 99: 115, 1932.
113. Nesbitt, H. T.: A Syndrome Occurring in the Neonatal Period Manifesting Tetany-like Symptoms, Am. J. Dis. Child. 44: 286, 1932.
114. Mitchel, A. G., and Stevenson, F. E.: Chvostek's Facialis Sign in New-born Infants, J. A. M. A. 99: 1502, 1932.
115. Von Reuss, A. R.: The Diseases of the Newborn, New York, 1921, William Wood & Company, p. 343.

9 Exchange Place

American Academy of Pediatrics

Proceedings

FIFTH ANNUAL MEETING OF THE AMERICAN ACADEMY OF PEDIATRICS

NEW YORK, JUNE 7 AND 8, 1935

The business meeting of the American Academy of Pediatrics was called to order at 10 A.M., June 8, 1935, at the Waldorf-Astoria Hotel, New York, N. Y., by the President, Thomas B. Cooley.

Report of the Executive Board of the American Academy of Pediatrics, Waldorf-Astoria Hotel, June 6, 1935

The report of audit was received and approved.

The statement of receipts and disbursements June 1, 1934, to June 1, 1935, was received and approved.

The proposed budget for the year June 1, 1935, to June 1, 1936, was received and approved.

The following were recommended for Emeritus Membership: A. B. Grossman, Cleveland, Ohio; Frank Leech, Washington, D. C.; Thomas C. McCleave, Berkeley, Calif.

The following were recommended for Associate Membership: William H. Park, New York, N. Y.; Mr. Perry Dunlap Smith, Winnetka, Ill.; Herbert R. Stolz, Oakland, Calif.

The Board recommends that Alfred A. Walker be reappointed as a member of the Board of Pediatrics.

At the meeting of the Executive Board in Evanston, Dec. 1, 1934, the following additions and changes in the Constitution and By-Laws were recommended:

ARTICLE III. Change title to "Powers and Duties of Executive Board, Regional Committees, and State and District Chairmen."

Add the following:

"Section 4. State, District or Provincial Chairmen. The President shall, on recommendation of the respective Regional Chairmen, appoint a Chairman for each state, district or province, as the case may be, in which the Academy is represented by Fellows.

"Section 5. The duties of each State, District, or Provincial Chairman shall be as follows:

"(a) To keep himself informed as to the purposes and policies of the Academy and as to conditions in his district relating to these policies.

"(b) To promote association and cooperation between the Fellows in his district, to consider with them the problems arising there, and to assist and initiate when necessary activities in furtherance of the Academy's aims and purposes."

ARTICLE III. Section 2. Shall be changed to read from "at least five members" to "three members."

The Board approved the report of the Committee on Scientific Policy of the Academy.

The Board approved the following report of the Nominating Committee:

Vice-President (President-Elect), Laurence R. DeBuys, New Orleans, La.

Chairman Region I, Louis C. Schroeder, New York City.

The Board recommends that there be created a Committee on Medical Legislation, one of the duties of which would be to investigate the possibilities of some action with regard to a National Department of Health.

The Secretary was authorized to formulate a plan relative to time and place of next meeting to be submitted to the Board for action by mail vote.

Report of the Secretary-Treasurer

On June 1, 1934, there were 607 Fellows in the Academy. At the Cleveland meeting 98 Fellows were accepted, and at the Executive Board meeting in December of last year 99 more. Five have been transferred to Emeritus Fellowship, and we have been unfortunate enough to have had four deaths. Our loss from death has included Dr. John Ruhräh, the much beloved ex-President of the Society and Dr. Roger Dennett, who has, since the Academy started, been active in its interests; and two recent members, Dr. S. Herman Lippitt of Milwaukee, Wis., and Dr. Charles F. Milligan of Clayton, N. M. The present Fellowship (May 1, 1935) of the Academy is 813, 5 Emeritus Fellows, and 13 Associate Fellows, making a total of 831.

The increase in the Fellowship, however, gives but little indication of the work the Society has been doing. This has increased tremendously in the last year. One can readily estimate the amount of work done by the fact that, whereas the expense for stamps in the Secretary's Office last year amounted to three hundred dollars, during this year the cost will be in the neighborhood of four hundred and fifty dollars.

We have gradually but surely systematized our regional work, and we have developed active State Chairmen in practically every state in which there is a Fellow. The Regional and State work is going out in nearly all the regions and states, and the special committees have been, in most instances, very active. The Committee on Child Health Relations with Dr. Martha Eliot as Chairman had a meeting in Washington, February 10, which all the members of the Committee and the Secretary attended. This was an all-day meeting and much was accomplished, as will be seen from the report from that Committee. The Committee on Clinical Investigation and Scientific Research has undertaken a very exhaustive investigation, which will be found in their report. The Committee on Cooperation with Nonmedical Groups and Societies has recently been appointed, but it is of some interest that we have established relationship with the National Society for Research in Child Development, with the Chairman of that Committee as chairman of the Pediatric Section and your Secretary as secretary. Dr. LeRoy A. Wilkes represented us at the annual meeting of the National Association for Nursery Education, and Dr. Edward L. Bauer at the Child Research Clinic of the Woods Schools of Langhorne, Pa.

The Committee on Hospitals and Dispensaries has completed its report on children's hospitals and pediatric departments of general hospitals, is collecting data on the contagious hospitals, and with the Committee on Medical Education has obtained data on residencies and interneships in pediatrics in this country. The Committee on Medical Education aside from the effort just mentioned has continued its efforts to raise the standard of pediatric teaching in this country which efforts we hope and trust will prove successful. The Committee on Nursing Education is perfecting a program as is the Committee on School Health and School Health Education.

The Secretary has attended, in addition to the meetings mentioned, a meeting of the State Chairmen and Regional Committee of Region II, at Nashville, Tenn., January 13, at which were discussed many of the general policies. Much of interest was brought out, and the State programs were crystallized.

Each of our regions now has a regional meeting. The Secretary was happy enough to be present at the one in New York, the first meeting of Region I, which was held October 19 and 20. This meeting was a great success and will be continued each year.

A change has been made in the general construction of the Academy, the whole being reduced to four regions, sections of Canada being divided between Regions I, III, and IV. This was at the suggestion of the Canadian members.

Our official Journal is fast becoming one of the outstanding pediatric journals in the world and is a great credit to its editors and to the Society as a whole. In other words, this Society has become the power in pediatrics in the United States. It has passed the period of formation, and we cannot expect that its membership will be greatly increased. We must now devote our efforts to organization of pediatric movements in the states and regions and cooperating with various national Societies which have to do with the care and health of children.

STATEMENT OF RECEIPTS AND DISBURSEMENTS
June 1, 1934 to May 15, 1935

Balance, June 1, 1934				$ 5,736.08
Receipts				
Initiation fees			$ 5,565.15	
Dues			12,126.00	
Exhibits			1,287.50	
Interest on bonds			300.00	
Premium on Canadian checks			2.10	19,280.75
				$25,016.83
Disbursements				
Office				
Salaries				
Secretary	$5,000.00			
Stenographer	1,560.00			
Office	367.50	$6,927.50		
Printing and stationery		537.56		
Postage		417.52		
Rent		240.00		
Office supplies		81.90		
Treasurer's bond		62.50		
Telephone and telegrams		40.01		
Exchange on checks		31.68		
Tax on checks		2.72		
Incidentals		140.54	8,481.93	
Purchase of tax warrants			3,093.56	
Subscriptions to JOURNAL OF PEDIATRICS			3,865.00	
Annual meeting			2,592.23	
Executive board meeting			612.68	
Traveling expense				
Secretary		270.44		
Special missions		279.20	549.64	
Regional expense				
Region I	266.43			
States:				
Maryland	1.53			
Massachusetts	18.45			
New Jersey	18.85	305.26		

Region II	79.27			
Meeting of State Chairmen	513.20			
State: North Carolina	2.00	594.47		
Region III	227.77			
States:				
Illinois	6.78			
Kansas	8.00			
Michigan	25.00			
Wisconsin	2.50	270.05		
Region IV	29.61			
State: Washington	4.59	34.20	1,203.98	
Miscellaneous committees				
Committee on Child Health Relations Meeting		576.96		
Committee on Clinical Investigation and Scientific Research		173.50		
Committee on Medical Education		5.70	756.25	21,155.27
Balance, May 15, 1935				$ 3,861.56

Assets as of May 15, 1935

Balance, May 15, 1935	$ 3,861.56
Savings account	3,490.13
$10,000 U. S. Treasury Certificates, 3 per cent, 6/15/35 ($100-31/32)	10,371.87
$3,000 Chicago Board of Education Building Fund 1934 Levy Tax Anticipation Warrants, 5 per cent ($101)	3,146.67
	$20,870.23

CLIFFORD G. GRULEE, Secretary-Treasurer.

Report of Region I

Region I, including the eastern provinces of Canada, now has a membership of 288, divided as follows:

Connecticut	17
Delaware	1
District of Columbia	12
Maine	5
Maryland	16
Massachusetts	36
New Hampshire	1
New Jersey	25
New York	101
Pennsylvania	57
Rhode Island	8
Vermont	2
Ontario	6
Quebec	1
Total	288

There has been a steady increase in interest as evidenced not only by the number of newly elected members, but by many inquiries. No changes of any great moment have taken place in the policies of the state committees, but those formerly outlined have been followed steadily.

There have been some inquiries relating to the policies of the Academy, which the Executive Committee is asked to consider. The first relates to the handicapped child. In some states and, possibly, under certain new laws which will

be passed at Washington, various sums of money will be allotted for the care of handicapped children. There has been a tendency in the past to think only of the child who is crippled as being handicapped. It would seem wiser to have a wider interpretation of the "handicapped child," so that public moneys, when distributed, may go to others than those who suffer crippling deformities.

This brings up the whole problem of chronic illness among children, because in the distribution of public moneys it would seem wise to formulate a policy so that those who suffer from any of the forms of chronic illnesses might benefit, as well as those who are crippled.

LOUIS C. SCHROEDER,
Regional Chairman.

Report of Region II

The new members admitted from June, 1934 to December, 1934, numbered fifteen, and the applications filed from January, 1935 to June, 1935, numbered eighteen.

Regional Items of Interest.—All states have been exceedingly active this last year. There were three changes made in the state chairmen:

Dr. W. W. Quillian, Florida, was appointed to succeed Dr. L. W. Holloway; Dr. E. G. Schwarz, Texas, was appointed to succeed Dr. J. H. Park, Jr.; Dr. J. L. Blanton, West Virginia, was appointed to succeed Dr. J. T. Thornton.

Every state in the South now has a pediatric society, or a pediatric section of the state medical society. Most of these societies are quite active and work in conjunction with the Academy.

The last state to organize was Arkansas. I wish particularly to commend the activities of the chairman, Dr. Morgan Smith. All pediatricians eligible are now enrolled in the Academy. A pediatric society, recently organized in Fort Smith, has approximately thirty members, admitting all men interested in child health problems who are members of the state society. Although this state was the last to organize, I believe it will make great progress as there is much enthusiasm, and all the members are working.

Dr. P. F. Barbour, chairman of Kentucky, is to be commended for the post-graduate work initiated in his state.

There will be no detailed report from each state as the activities will be taken up in the Journal later.

In a recent survey made of the Southern states, it was shown that four-fifths of those men eligible are now enrolled in the Academy. If next year's activities equal those of the past year, we will have 100 per cent enrolled.

Region II met in San Antonio, Texas, last November. The program consisted of four round table discussions. Preparations were made for fifty men at each table though more than one hundred were present at some of the tables and many were turned away for lack of space. This meeting was open to all members of the Southern Medical Association. Our business meeting was well attended. The Council was represented by Dr. Thomas B. Cooley, our president. Next year there will be a joint session with Region III in St. Louis at the same time as the meeting of the Southern Medical Association. This joint meeting will give us an opportunity to renew old friendships formed in the Central States Pediatric Society. A full attendance from this region is urged.

On January 13 a meeting of the state chairmen of this Region was held in Nashville. Dr. Clifford G. Grulee represented the Academy, and Dr. Martha Eliot, Chairman of the Committee on Child Health Relations, was present. There was a full discussion of the general policies of the Academy and its purpose. There was a report from each state. All chairmen were present except from Texas,

Oklahoma, and West Virginia. Dr. Eliot outlined the plan and purpose of the Committee on Child Health Relations. It was decided at this meeting that each state would adopt a plan similar to the one outlined by Dr. Eliot, adapting it to the needs of the particular state. The purpose was to give every member of the Academy in this region something to do, thereby obtaining the interest of each individual member. You will all be called on at some time during the year and we hope the efforts of the state chairmen will meet with a ready response.

I wish to take this opportunity to thank the regional committee, the state chairmen, and the members of the Academy in this region for their loyal support during the past year.

EDWARD C. MITCHELL,
Regional Chairman.

Report of Region III

The membership of Region III as of January 1, 1935, was 283. This represents the great majority of pediatricians of the region. At the recent meeting of the Regional Committee on April 27, 1935, fifteen additional men were recommended for membership to the Council. There are still a small number of men in various states who should be members of the Academy, many of whom have not joined for financial reasons. It is hoped that most of them will join in the near future. Since over half of the present applications pending have come in through the activity of one state chairman, I feel sure that by similar efforts of the other twelve state chairmen, our membership can be increased to include practically all the qualified men and women in the region.

It is becoming more evident each year that the active work of the Academy will be carried on by state organization within the region and that a committee of state chairmen rather than the Regional Committee will become the dominant group. During the past year, the following new chairmen have been appointed: Dr. E. J. Huenekens, Minnesota; Dr. H. C. Berger, Missouri; and Dr. S. H. Ashmun, Ohio. I regret to report that within the week Dr. Bernard Carey has resigned the state chairmanship of Michigan. Dr. Carey has done an excellent piece of work, and his influence will be missed.

The outstanding event of the year was our clinical meeting held in Rochester and Minneapolis on October 4 to 6. Representation from Ohio, Kansas, and Missouri was reduced by two conflicts, with the state meeting in Ohio and the clinical meeting in Kansas City. Invitations to the meeting were sent to the pediatricians in Manitoba through Dr. Chown. A number of men from Canada were in attendance.

The new feature that we introduced into the program was a talk on a topic allied to pediatrics. Mr. Perry D. Smith, Headmaster of the Winnetka County Day School, gave a very interesting address, "What Is Good Practice in Modern Education (Pediatrician and the Pedagogue)?"

A large number of members of the old Central States Pediatric Society attended the meeting. Invitations were sent to all who were members at the time of the disbanding of that Society. I obtained from all but three essayists short abstracts of their papers. These have been published in the JOURNAL OF PEDIATRICS in the form much as were the Proceedings of the Central States Pediatric Society. The address by Mr. Perry Smith appeared in the March issue of the JOURNAL OF PEDIATRICS."

Preceding the scientific session, the business meeting of Region III was held in the morning of the last day. The order of business was as follows: reports

of state chairmen, new business, and election of officers—chairman of clinical meeting, vice chairman, and secretary and editor.

The reports of the various state chairmen were presented and were published in the JOURNAL OF PEDIATRICS. Dr. A. Graeme Mitchell was elected chairman for 1935, and the next meeting place was to have been Cincinnati. In view of the Southern Medical Association meeting in St. Louis this year, the Regional Committee was unanimous in postponing the Cincinnati meeting for one year and having a joint meeting with Region II in St. Louis this fall. The Regional Committee appointed Dr. T. C. Hempelmann, chairman; Dr. Gustave Lippmann, vice chairman; and Dr. A. F. Hartmann, secretary and editor. The meeting is to be held the week-end before the meeting of the Southern Medical Association in November.

In order to emphasize the importance of the state work, a luncheon was held, at which practically all the states were represented. After considerable discussion of state programs, a committee of four was appointed consisting of the chairmen of Michigan, Indiana, Iowa, and Illinois with Dr. Maurice Blatt, of Chicago, as chairman. This committee is to draw up a complete state program and report at the next meeting.

In the recent reports from state chairmen, the following points were emphasized: (1) the importance of presenting pediatric papers before general sessions of state societies, (2) closer cooperation between pediatrician and state boards of health, (3) cooperation with state and county relief organizations, and (4) the initiation of tuberculosis surveys by local medical societies. One state group requested a special discussion of medical economics at the next annual meeting.

The following suggestions were made to the Regional Committee for action by the Academy: the supervision of summer camps and the control of radio advertising.

H. F. HELMHOLZ,
Regional Chairman.

Report of Region IV

In Region IV Academy activities have gathered momentum and the membership has increased until at present a very large proportion of those who devote themselves entirely to pediatrics are actively identified with the organization. In certain western states where only a few are eligible for membership the Academy is fortunate in having representatives of extremely great zeal and energy. In Idaho, Dr. Harmon Tremaine, the only member in the state, has organized and carried forward during the past two years state-wide child health activities, public health nursing, and a campaign for immunization against communicable diseases. In New Mexico, Dr. Meldrum Wylder reports the organization of prenatal and postnatal work carried out by public health nurses secured by the aid of the F. E. R. A. There has been a perceptible improvement in infant mortality and a reduction in summer diarrheas as a result. The untimely death of Dr. C. F. Milligan is to be regretted. Dr. Milligan was president of the New Mexico Medical Society and was most active in public health pediatrics. In Arizona, with only two members, a survey of child welfare organizations is being carried out by the state chairman, Dr. Vivian Tappan.

In more populous districts Academy activities have proceeded along with those of preexisting organizations. In Utah the membership is identified with the very active Intermountain Pediatric Society. In the Pacific Northwest, the Northwest Pediatric Society has undertaken preschool examinations and has conducted pediatric clinics for the instruction of the profession at large. The Southwest Pediatric Society, including Los Angeles, San Diego, and surrounding area, is largely composed of Academy men. Their most important contribution has been

their successful effort to secure the inclusion of health supervision and preventive measures under the term of "medical care" in the state health insurance measure under consideration by the legislature at the present time and to insure proper inclusion of the pediatrician in this measure by suggesting the substitution of the term "any licensed physician" for the term "general practitioner" in the bill.

In the San Francisco region the old Northern California Pediatric Society has been entirely disbanded and in its stead has been developed a local "chapter" of the Academy. Numerous meetings have been held during the past year, some of them essentially practical, and others more purely scientific. Excellent cooperation from the local Board of Health has been secured through the Director, Dr. J. C. Geiger, and his associates, Dr. George H. Becker and Dr. Paul S. Barrett, in outlining plans for communicable disease control, immunization, preschool and school health examinations. Dr. C. A. Aldrich addressed a large meeting of the membership and many guests in February on the subject, "Nephritis in Childhood." A later meeting considered a report on the supervision and nutrition of children in various institutions, orphanages, day nurseries and kindergartens in San Francisco.

The Regional Meeting was held in Oakland, Calif., October 26 and 27, 1934, the scientific program being under the direction of Dr. Clifford Sweet. There was a large and enthusiastic attendance from the entire region, and a most stimulating program was presented. The next regional meeting will be held in Seattle, Wash., August 9 and 10, 1935, the scientific program being arranged by Dr. Jay I. Durand.

EDWARD B. SHAW,
Regional Chairman.

Report of the Committee on Child Health Relations

The Committee on Child Health Relations has held two meetings during the past year. At the first one held in Cleveland, on June 10, 1934 (present, Dr. Barbour, Dr. Crawford, Dr. Nichols, and Dr. Eliot), the function of the committee and the program for the coming year were discussed at some length, and two recommendations to the Executive Board of the Academy were adopted: the first having to do with the change of the Committee's name from the "Committee on Relation of the Academy to Philanthropic Agencies, Welfare Workers, Public Health Agencies, etc." to the "Committee on Child Health Relations"; and the second with regard to the desirability of having state groups inform themselves as to the facilities for health supervision and medical care of children in their respective states with a view to improving conditions where necessary. The Committee suggested also that it prepare an outline for a state-wide appraisal that would assist state groups in selecting the phase or phases of child health work most in need of study. A subcommittee of two (Dr. Crawford and Dr. Eliot) was appointed to draw up a preliminary draft of such an outline for presentation to the Committee at a midwinter meeting. The recommendations of the Committee were forwarded to the Executive Board and after some correspondence with the Committee were approved by the Board in the following form:

1. That the name of the committee be changed to the Committee on Child Health Relations.
2. That, wherever feasible, the state Academy groups undertake to inform themselves as to

(a) the status of child health activities carried on in their respective states by official and nonofficial agencies;
(b) the provisions for medical care and health supervision of children in institutions or under the care of child-placing agencies and of children in their own homes whose families are unable to pay for medical service;
(c) the medical and health needs of special groups of children scattered throughout the state or concentrated in certain areas or communities; and
(d) the basic facts of mortality and morbidity among children in the state and its political subdivisions

for the purpose of appraising the work done and of bringing about improvement in the health supervision and medical care of children through the cooperation of official departments of health and welfare, medical societies, nonofficial welfare agencies, and lay organizations.

The Committee on Child Health Relations would be glad to attempt the preparation of a brief outline giving suggestions for such an appraisal of child health activities if state groups desire it. The Committee appreciates that certain state groups are already engaged in appraising and developing child health activities and does not intend to suggest any duplication of effort.

At the suggestion of the Chairman two new members were added to the Committee in December, 1934, Dr. George M. Lyon, of Huntington, W. Va., and Dr. M. Hines Roberts, of Atlanta, Ga.

Following the approval of these recommendations, the Committee held a midwinter meeting on February 10, 1935, in Washington, D. C. (present, Dr. Barbour, Dr. Crawford, Dr. Lyon, Dr. Nichols, Dr. Reiss, Dr. Roberts, Dr. Eliot, and Dr. Grulee), to consider "A Suggested Outline of Appraisal of Facilities for Health Supervision and Medical Care of Children." After an all-day discussion of the details of the drafts presented by the subcommittee, certain recommendations with regard to its form and content were made, and it was voted that copies should be submitted to state chairmen just before the Academy meeting in June, 1935. (The outline will be published in a later issue of the JOURNAL.)

It was voted and approved by Dr. Grulee that the Committee be given time at the Academy meeting in June to discuss with the state chairmen the suggested outline of appraisal. It was voted that if such a meeting could be held, certain members of the Committee should present designated major phases of the problem as follows:

Dr. Crawford: "A method of state-wide study of health supervision and medical care given to children in child-caring institutions and the physician's responsibility in directing health and welfare activities in such institutions."

Dr. Lyon: "Infant and maternal mortality statistics."

Dr. Nichols: "The need for postgraduate education of general practitioners in the field of child health."

Dr. Eliot: "The use of the suggested appraisal outline and the need for a close relationship between state health departments and the state Academy groups."

Dr. Grulee agreed to try to arrange such a meeting of state chairmen for Friday evening, June 7.

The Committee also has under consideration the advisability of recommending to state chairmen the formation of (1) state child health advisory committees which should include representatives of various public and private (official and nonofficial), professional and lay organizations that have an interest in child

and maternal health, or (2) of state-wide councils on child health and welfare, which should include representatives from public and private, professional and lay organizations that have an interest in child welfare in its broadest social and economic sense as well as those organizations that have an interest in child and maternal health. It was the consensus of opinion in the committee that the latter type of council would have a broader scope of interest since it would include all problems of child life in its deliberations, but in the case of some states it was thought that the development of a state child health advisory committee might be a necessary first step toward the formation of a child council. No special recommendation was made to the Executive Board of the Academy in this respect at this time.

MARTHA M. ELIOT,
Chairman.

Report of the Committee on Cooperation With Nonmedical Groups and Societies

The Committee on Cooperation with Nonmedical Groups and Societies wishes at this time merely to report progress. It has been impossible to have a full meeting of the Committee since its appointment, but preliminary work has been done in a listing of those national organizations which the Committee should contact. The Committee will have its first meeting at the Annual Meeting in New York when it is hoped additional members will be added to the Committee and a definite mode of procedure decided upon.

HENRY F. HELMHOLZ,
Chairman.

Report of Committee on Hospitals and Dispensaries

During the past year, the Committee on Hospitals and Dispensaries has completed and published detailed reports concerning thirty-eight hospitals in the United States and Canada which can be classified as exclusively children's hospitals. Similar reports have been made concerning the pediatric departments of all general hospitals in the United States which have twenty-four or more beds and bassinets for infants and children. Detailed comprehensive information was secured through the use of questionnaires, and all departments of the hospitals were investigated. These reports have appeared at intervals in current issues of the JOURNAL OF PEDIATRICS and the *Bulletin of the American Hospital Association.*

At the present time, the Committee is collecting similar information concerning contagious disease hospitals and contagious disease departments of general hospitals. These reports will appear in due time in the same journals.

In the near future the Committee, in conjunction with the Committee on Medical Education, will compile and publish a pamphlet which will contain pertinent information concerning pediatric residencies and interneships in all hospitals of the United States and Canada.

The Committee wishes at this time to thank all members of this Society who have so efficiently cooperated with us to secure the information we have sought.

GEORGE F. MUNNS,
Secretary of the Committee.

Report of the Committee on Medical Education for 1935

In the past year a survey of teaching hours in pediatrics has been made for the purpose of comparison with the survey of the Third White House Conference report five years previously.

It is exceedingly difficult to make generalized comparisons of value as such marked differences in the utilization of hours exist. There is an increasing use of clinical clerkships by which the student works in the wards for "one week" or "one month."

Tables I, II, and III show a definite trend in the last five years to allot an increasing number of hours in the medical school curriculum to pediatrics. A comparison of required hours in forty-two schools, in instances in which the survey figures are complete for both periods, shows the average has increased from 176 in 1929 to 214 in 1934-35. As will be noted, many schools give elective courses in addition to the required hours.

TABLE I

REQUIRED HOURS IN PEDIATRICS IN 42 MEDICAL SCHOOLS

	1929-30	1934-35	PLUS OR MINUS		1929-30	1934-35	PLUS OR MINUS
1*	135	189	+ 54	29	213	321	+108
2*	240	272	+ 32	32	122	136	+ 14
4*	223	344	+121	35*	178	201	+ 23
5	437	420	− 17	36	192	252	+ 60
6*	88	176	+ 88	37	162	220	+ 58
7*	142	154	+ 12	38	180	171	− 9
8*	111	108	− 3	40	40	180	+140
10*	218	240	+ 22	41	175	144	− 31
11	160	160	+ 9	44	239	350	+111
12*	198	205	+ 7	46	104	186	+ 82
13*	148	176	+ 28	47	120	182	+ 62
14*	146	112	− 34	48	112	173	+ 61
15*	144	166	+ 22	51	211	211	0
16*	298	369	+ 71	52	156	270	+114
17	264	312	+ 48	53	166	201	+ 35
18*	223	321	+ 98	54	208	206	− 2
19*	154	154	0	55	80	131	+ 51
20*	170	165	− 5	56	104	112	+ 8
24*	132	149	+ 17	59*	90	215	+125
25	312	316	+ 4	60	189	225	+ 36
27	266	260	− 10	61	154	196	+ 42

*School in addition to these required hours has elective courses in pediatrics.

The number of the school is the number given in the Third White House Conference report. Schools are not listed alphabetically.

TABLE II

SUMMARY OF CHANGES IN HOURS 1929-30 TO 1934-35

	NO. SCHOOLS		NO. SCHOOLS
Decrease over 10 hours	3	Decrease 23%	1
Decrease 1 to 10 hours	5	Decrease 17½%	1
No change	2	Decrease 1 to 5%	6
Increase 1 to 10 hours	4	No change	2
Increase 10 to 25 hours	6	Increase 1 to 10%	5
Increase 25 to 50 hours	6	Increase 10 to 15%	6
Increase 50 to 75 hours	7	Increase 15 to 25%	5
Increase 75 to 100 hours	3	Increase 30 to 40%	3
Increase over 100 hours	6	Increase 40 to 50%	4
		Increase 50 to 60%	4
		Increase 70 to 80%	2
		Increase 100%	1
		Increase 140%	1
		Increase over 300%	1

The committee report of the Third White House Conference agreed upon 200 hours as approximately the minimum number of hours necessary for adequate undergraduate pediatric instruction.

Table II shows that the percentage of schools with less than 200 teaching hours has fallen from 70 to 50. Even where elective undergraduate courses are offered, the trend has been to increase required hours. In Table III a decided "shift to the right" is noted for the 1934-35 figures. While in 1929-30 sixteen of the forty-two schools were allotted less than 150 hours, in 1934-35 the number has decreased to seven. The number of schools with over 300 required hours increased from two to eight.

TABLE III

SUMMARY OF REQUIRED HOURS IN 42 SCHOOLS

	UNDER 100	100-150	150-175	175-200	200-225	225-300	OVER 300
1929-30	4	12	7	6	6	5	2
1934-35	0	7	9	5	7	6	8

	1929-30	1934-35
Average required hours	176	214
Schools under 200 required hours	70%	50%
Schools with inadequate hours (under 150)	38%	16%

The question of specialization has been dropped as an activity of the Committee on Medical Education since the Academy joined in the formation of the American Board of Pediatrics which has been actively at work this past year.

One of the most important problems at present is the matter of opportunities for training open to physicians planning to specialize in pediatrics. After conferences with the chairman of the Committee, the Academy Committee on Hospitals has collected detailed information of the available house officer positions in pediatric clinics which have at least a twelve-month service. Such information is being tabulated and should be of great value to younger men considering taking up the study of pediatrics as a specialty.

BORDEN S. VEEDER,
Chairman.

Report of the Committee on Mental Hygiene

The Committee on Mental Hygiene has either a very easy or an almost overwhelmingly difficult job. It is easy if it simply emphasizes the duty of the pediatrician to enlarge his professional horizon and to care for "the whole child." It is less easy to suggest changes in pediatric practice.

Some of the members of the Academy must remember with vividness the remarks of Dr. William Blatz, of Toronto, who found pediatricians less aware of certain phases of psychology than the average mother trained by him. Others must have faced scorn of the same sort from other specialists.

The attempt to resolve all the difficulties about psychology and psychiatry by claiming precedence for pediatrics in every situation in which children are involved is quite obviously going to lead us into ridiculous situations, from which we can escape only by emphasizing medical prestige and hiding behind it.

Your committee believes that a majority of the teachers of pediatrics are trying their best to find a practicable way out of the confusion. The various attempts are of interest.

The method of introducing a fully equipped and conventional guidance clinic into the teaching pediatric clinic is being tried in some places. The hope, of course, is that students, internes and the staff will gain an understanding of the methods used. Certain obvious difficulties exist. The children under review are usually free from serious physical disease. The time required for adequate appraisal is so great that relatively few students or internes see end-results. Unless genuine and insistent interest can be maintained, the formal clinic is likely to become isolated.

In other places more or less adequate consultation is available. The effectiveness of this relationship is, of course, dependent on the ability and tolerance of the doctors concerned.

At one or two schools, the challenge of growth and development has been more or less completely met by the establishment of special clinics with highly competent staffs. In these clinics the psychologic approach is emphasized. Again one difficulty occurs: the children are not sick or under ordinary pediatric supervision.

Another method which seems worth trying is to see what happens when the pediatrician selects "psychiatric" or "psychologic" material and tries to deal with it with the help, but not the direction, of others.

None of these methods have been tried out over any long period, but they are all worth watching.

Your Committee ventures the following remarks:

1. The pediatrician, as he stands, is not cooperating in any effective way with the groups carrying on mental hygiene activities.

2. It is by no means self-evident that, without cooperation, he can take satisfactory care of an important group of patients.

3. The present situation is likely to persist until attention to mental and emotional difficulties is emphasized in pediatric teaching and until pediatricians find methods of dealing with appropriate problems.

4. The first step is to study the problems that arise in direct sequence to physical disease. There is no question that the doctor must remain in charge of this group.

5. Except for this group, the question arises as to control. Doctors should be very careful about claiming control of "the whole child" and should welcome discussions with psychiatrists, psychologists, social workers, and teachers. Out of such discussion come suggestions which will help any given doctor to find his place in a mental hygiene program in his own community.

6. The pediatricians will, in the opinion of this committee, be fulfilling their obligations whenever they organize their training and their practice so that the children under their care receive prompt and intelligent attention to the intellectual and emotional difficulties which arise. It is not at all certain that the practicing physician can provide all this care himself, but he should use his influence to see that adequate care is made available and that parents use it wisely.

7. Any plan which is suggested for pediatric training should be subjected to very careful study. The fundamental duty of the pediatrician is to be a skillful and available supervisor of the physical status of the well child and a competent and available doctor for the ill child. Any responsibility for the control of mental or emotional difficulties can be accepted only if it does not impair his primary responsibility. The "whole child" is a term which must be defined by the pediatrician himself.

BRONSON CROTHERS,
Chairman.

Report of Committee on School Health and School Health Education

I have corresponded with a number of the leading nurses in the field of nursing education. The general feeling is that there has not been enough emphasis on the training for the well child. This is particularly true from their own statements in public health work. It is very apparent in institutional work and private nursing. The nurse has never been taught anything about the well child, what he is like, and has little or no knowledge of the standards of mental, social, and emotional growth and how these are reflected in the child's behavior. More training along these lines would prevent the many mistakes which the nurse makes in discussion with mothers and the advice which she often inadvertently or unintentionally gives when discussing the child, not that the nurse is trying to prescribe, but it often is impossible not to answer questions which the mother asks. Also, she is handicapped in the method she uses in handling the child whether sick or well.

The National League of Nursing Education at their last meeting formed a committee on the child; the object of this committee being to bring into the education of the nurse some of the important knowledge in child development. I am in correspondence with this committee, and I think the cooperation between the Committee on Nursing Education in Pediatrics and this special committee on child development of the National League of Nursing Education will do a good deal for the problem of nursing in pediatrics.

WILLIAM PALMER LUCAS,
Chairman.

Report of Committee on School Health and School Health Education

A survey made of the available reports and of opinions of selected leaders engaged in public health administration, school medical inspection, and private practice indicates that there is a wide diversion of activity now being conducted in the various parts of the country, and that there is no evidence that these practices are efficient or economical in use of time and personnel, including the physicians.

A questionnaire was submitted to thirty of the country's leading public health administrators and physicians, long-experienced in school health work. The replies show rather general agreement on the questions submitted:

1. "Is it economically possible to give a real periodic health examination in the school?"

There was an almost unanimous agreement that it is not the purpose of the schools to provide real periodic health examinations, desirable as these may be from the standpoint of each individual pupil. The inspection for communicable diseases and for real handicaps which interfere with education are justifiable and within the economic limitations allowable for health services placed in any educational program.

2. "Is the work now being done in public schools a worthy example of real medical service?"

The preponderant opinion was that it is not educationally sound, in that it does not represent typical medical service such as can be found in hospitals and in the private office of the physician. Therefore, it is unsound to hold this out educationally as an example of something which it does not truthfully represent.

3. "Is this not bad for an educational viewpoint?"

Again the preponderant opinion was that it was not good education because it did not represent medical service at its best, and also because parents looked down on the physician in the school when he conflicted in any way with his

colleague, the family doctor. This probably accounts for the low percentage of reported defects which are actually corrected, even under the pressure and urge of the school-teacher and the nurse.

4. "Is the average school physician competent and prepared to pass upon the soundness of the subject matter used in health education?"

The agreement of this question was preponderantly that he is not. The average physician engaged in school work does not keep up with the latest improvements in medicine and does not read extensively, nor is his practice extensive enough to provide him with authority to pronounce what represents the latest approved technic in medicine or thought in health knowledge.

The question of intervals between examinations which should be recommended is a problem which is not yet solved. All interviewed on this question were agreed that the yearly interval is too frequent but are not yet in a position to state definitely what that interval should be. There seems to be quite a common agreement, however, that there should be an examination on entrance to school and on entrance to high school. A number proposed examination just prior to leaving school for the purpose of going to work. Some suggest a recheck in the third grade, but all are quite agreed that too many "potential" defects are being recorded and that these are called to the attention of parents as urgent, thereby causing unnecessary conflict in medical opinion, between the school physician, who makes the original inspection, and the family physician, who more carefully, leisurely, and thoroughly follows up on the examination and makes the final decision.

For instance, in the study made by the American Child Health Association for the Metropolitan Life Insurance Company of New York City recently, it was reported that over 60 per cent of the children in the elementary grades have had their tonsils removed. In the literature of the last two or three years from England, there has been considerable discussion over the high percentage of tonsillectomies performed on school children in that country, reported to reach 22 per cent. In Germany, there is a report that the average percentage of children having tonsillectomies performed upon them has reached the total of 5 per cent. Such discrepancies show a lack of agreement in professional opinion as to the necessity for this operation. School examiners' figures vary greatly on the subject depending upon the examiner, and even in the same examiner on different occasions. From such wide variation of opinion, it is probably best to report only extreme cases until we have more agreement on the others.

The result of the survey was certainly convincing as to one thing. There is considerable room for improvement in the study of the physical condition of the children now attending school. There seems to be a general opinion that the work should be directed toward the earlier levels and that children should be properly prepared by the physicians, at the parents' request, in the preschool age period instead of waiting until after they enter school. Immunization against disease should be fully taken care of before the child enters school, since more than one-half of the children who die from diphtheria die before they enter school for the first time.

The principal recommendation of the Committee at this time is that children in the preschool age receive more careful medical supervision, with the idea of sending them to school fit to profit most from the educational opportunities offered in the schools and without loss of time which should be devoted to educational purposes.

In order to carry out this recommendation that the child be prepared before entering school, it is necessary that some organized effort be made whereby

private physicians will provide a practical program that will take care in all economic levels of these children. It is suggested that the county medical societies and the state medical society, of which they are component units, be organized under a Public Health Committee to take charge of the plan for providing this opportunity for preschool age supervision for those who cannot afford to pay, as well as for those who can, so that the school may be furnished with a reliable record that will give them a very definite idea as to the physical qualifications, and possibly classification of the children who are entering school.

If any of the defects which are uncorrected when the child enters school, are significant enough to warrant the time and effort involved, the school nurse or the district nurse should follow up to interpret to the parent in the home these defects and the need for prompt medical attention for them by the private physician. She should be able to announce that this medical care is available in all economic levels and should be very specific as to the arrangements and procedure by which those indigents may be able to receive the care which she recommends. Adequate provision must, of course, be made for all, especially the Negro in the South.

In the survey mentioned, there was general agreement that all work done in the schools must be justified on an educational basis, and that only so far as the medical service was a direct and necessary aid in educational effort should it be justified and paid for out of school funds. It should not be forgotten that there are other agencies in the community which were founded primarily for health service. Upon these the schools should call for aid in solving the health implication in their work. There is no justification for a school to provide a duplication of the work of these agencies, when mutual arrangements of two existing agencies in any community can provide the best solution.

It is interesting to know that in America at least the foundation of a school medical inspection was specifically to control the interference with education caused by communicable disease and by those defects which actually interfered with educational progress.

So long as these aims were kept clearly in mind and the procedure was restricted to these activities, there was no criticism. It is the opinion of the Committee that the present need is not for more inspectors and nurses in the schools, but for a clarification of the aims of the schools and a plan by which the medical personnel could aid in the educational activities as medical advisors to the schools, with the actual service element being carried on through cooperation of the medical society. The medical advisor engaged by the school should act as the liaison officer between these two community groups in a mutually beneficial and cooperative community project.

This supplementary report of the Committee is to be added to the report submitted last year, as it is in direct conformity with that report, and, while it has not to date reached the full scope of the study as proposed, it is an evidence of the fact that the proposed study was laid out along the right lines because the supportive opinion and observation has subsequently been made in the interval and supports the last report.

The subject of medical inspection and general health study in the schools is an enormous subject involving a large amount of money and considerable personnel. It is worthy of continued study by the Committee for a number of years. It is the regret of the present Chairman that other duties recently assumed have made it impossible for him to carry on further as Chairman. He requests that another member of the Committee be made Chairman. If it is the wish of the Academy that he continue as a member of the Committee, he will be glad to do so, but he feels that in the capable hands of a man who is well fitted and

has the time to devote to it, progress can be made faster. Dr. Harold Mitchell is respectfully suggested as a man so equipped and who has the time to contribute more to the Committee than the present Chairman can at this time. His name is proposed as the Chairman of the Committee by the present Chairman.

The material accumulated by the present Chairman will be gladly turned over to the New Chairman so that the program already made can be utilized and added to by the new Chairman.

It is proposed that at this time the Committee concentrate on correct principles for the establishment of future school health work as it relates to the medical profession. Until the future plan of medical practice shall be definitely established, no marked change in the procedure in any given place need be made, unless it is not in accord with the principles announced.

LeRoy A. Wilkes,
Chairman.

Report of the Committee on Clinical Investigation and Scientific Research

The foregoing investigation of the Steenbock patents has drawn the attention of this Committee to a practice which has become increasingly prevalent in late years—that of commercial concerns making research grants to particular individuals and institutions for the study of a therapeutic agent or for a related problem. Such grants are usually made to individuals and institutions whose integrity is beyond question, and the terms of the grant are such that the grantee is allowed complete freedom of publication in regard to the results obtained. In spite of these safeguards this Committee wishes to point out that there are certain dangers inherent in the present practice and to offer a suggestion for remedying them.

From conversations with manufacturers it is clear that such grants are usually made in the hope that at least part of the research will yield results of commercial value to the company though it is expected that results of scientific value unrelated to the interests of the company will also be achieved. An analysis of publications made under such grants indicates an unusually high percentage of reports favoring the product of the manufacturer making the grant. We wish to point out that this result does not imply intellectual dishonesty on the part of the recipient of the grant. There are several possible explanations. This recipient may elect to receive grants only in regard to products about which he has a preconceived favorable opinion. There can be no doubt, however, that it is only human nature to be kindly disposed toward those from whom benefits are received and that even though not stated a moral obligation is assumed by the recipient of a grant, provided this does not conflict with other moral obligations already assumed. Such a moral obligation can be fulfilled in a variety of ways without intellectual dishonesty. Results favorable to the company can be published with more alacrity than those which turn out to the contrary. Researches which appear to be yielding products of value to the grantor may be pushed with more enthusiasm than those which turn out contrariwise. In the published work there is the constant temptation to deal more gently with one's friends than with one's enemies. This situation we believe to be undesirable.

It seems altogether desirable that manufacturers of foods and pharmaceuticals used by the pediatrician should contribute to pediatric research. They share with the pediatric research worker the objective of discovering new products of value and of revaluing old products. It is to their interest to have claims for the value of their products based on sound fact. It is our belief that manufacturers will still be willing to contribute toward sound pediatric research, even at the

expense of foregoing that peculiar good will and favorable prejudice which they obtain under the present system of making grants.

It is the opinion of this Committee that a national organization such as the Academy or the National Research Council with pediatric representation could obtain funds from manufacturers to study various agents about whose potency and value considerable doubt now exists; that the Academy could then dispense such funds to competent investigators who would then be under no moral obligation except to the Academy itself.

A number of examples could be cited of problems of interest to pediatricians as a whole for the solution of which commercial concerns might be induced to contribute collectively. One of the most obvious of these is an accurate comparison of the potency of the various "vitamin D fortified" milks in man. A letter written to the Academy by Dr. Lafayette B. Mendel expressed the hope that the Academy would sponsor a thorough investigation of this problem. Another problem that comes to mind, upon which pediatricians are still much confused, concerns the merits of the various sugars used in infant feeding. It seems possible that the manufacturers of various sugars would contribute to an investigation sufficiently thorough to settle this question once for all.

It may be pointed out that a similar policy is now being carried out by the National Research Council which has collected funds from a number of pharmaceutical houses for a series of investigations designed to develop and test a non-habit-forming narcotic.

It is obvious that such a plan can be successful only if the distribution of funds for research projects is carried out on the strictest principles without regard to considerations of personal friendship, geography, or medical politics. Such grants should be given only to investigators who have proved their ability to carry on work of the quality desired.

It is, therefore, suggested by this Committee that the Academy consider the advisability of pursuing the policy outlined above.

FRITZ B. TALBOT,
Chairman.

Annual Report of the American Board of Pediatrics, Inc., to the American Academy of Pediatrics

The American Board of Pediatrics wishes to make the following report. All told, 429 men have applied to the Board for certification. Of these, 339 have been certified. The rest are still under advisement. Sixty men have been examined, of whom fifteen failed. It may be of interest to the American Academy of Pediatrics that 273 of the members of the Society have, up to date, applied for certification.

Needless to say, the Board does not feel that it can add anything to the reputations of the men who belong to the American Academy of Pediatrics by issuing certificates to them, but on the other hand it does feel that the fact that members of this Society apply for certification will make the Board's certificate of more value to the younger men for whom this machinery is set up.

The members of the Board have had to stand for a certain amount of criticism, and, as the Board functions in part as an agent for the American Academy of Pediatrics, it feels it has the right to ask for the support of its members.

BORDEN S. VEEDER.

The Secretary asked for a discussion on the finances of the Academy. This request was granted, and an abstract of the discussion will be sent to each member of the Society.

The Secretary announced that in addition to the deaths already recorded, those of Mark Jampolis, of Chicago, and Joseph R. Shuman, of Los Angeles, should be noted. The members rose in tribute to those who died during the year.

There was some discussion on the Report on the Scientific Policy of the Academy. The report of the Committee on Clinical Investigation and Scientific Research was approved.

Dr. Crothers wished to add to his report the fact that we must recognize that the pediatrist has nothing to do with adults, and therefore his function does not involve the actual psychiatric care of the adult.

The report of the Executive Board was approved throughout including the recommendations for Emeritus Fellowship, Associate Fellowship, Active Fellows and the reappointment of Dr. Alfred A. Walker, of Birmingham, Ala., as a member of the Board of Pediatrics, the change in the Constitution and By-Laws, and the recommendation that a Committee on Medical Legislation be created, "one of the duties of which would be to investigate the possibilities of some action with regard to a National Department of Health."

Dr. Laurence R. DeBuys, of New Orleans, was elected Vice-President (President-elect) and Dr. Louis C. Schroeder, of New York City, was reelected Chairman of Region I.

Dr. Cooley then released the chair to Dr. Henry Dietrich, of Los Angeles, who made the following remarks:

"Gentlemen: I come before you in a very humble spirit. I am proud to occupy the presidency of this Academy. However, when I think of my predecessors in this office and what their names and accomplishments have meant to medicine, and particularly pediatrics in this country, I am more than overcome with a sense of humility. Dr. Ruhräh, in his presidential address, said, 'There is work for everyone; will we do it?' When one views the attendance at this meeting and the enthusiasm in the various committee meetings, the expression of desire regarding the various questions which have been discussed this morning, I think one cannot help but feel convinced that this organization and every individual member of it is ready and willing to do his part. I want to express to you my heartfelt thanks for your good will and expression of confidence. I thank you."

The meeting adjourned at 11:30 A.M.

FOURTH ANNUAL MEETING OF THE AMERICAN ACADEMY OF PEDIATRICS

CLEVELAND, JUNE 11 AND 12, 1934

Round Table Discussion on the Tonsil Question

Leader: Dr. Isaac A. Abt, Chicago

The Round Table Discussion on the Tonsil Question, held in connection with the American Academy of Pediatrics Fourth Annual Meeting of June 11 and 12 at Wade Park Manor Hotel, Cleveland, Ohio, was called to order by Dr. Isaac A. Abt, Chairman.

General Considerations

DR. A. D. KAISER (ROCHESTER, N. Y.).—Progress in the knowledge of the tonsils has been stationary for the past ten years. My interest in the question resulted from a study of rheumatic fever. Recurrent infections of rheumatic disease are often due to residual tonsillar infection.

From a historical viewpoint, tonsillectomy is an ancient operation since it was performed 2,000 years ago. The operation consisted in tying a cord around the protruding portion of the tonsil and letting it slough off and in that way getting rid of the surplus tonsil.

It was not until about a hundred years ago that any new information on the tonsil could be found, and then the operation was one largely of removing the surplus tissue. The real incentive to the study of the tonsils came at the time when the theory of focal infection was rather popular. In the course of a very few years, dating back to about 1910 or 1912, the tonsil received perhaps an undue amount of prominence as a focus of infection. Tonsillectomy became a popular fad. In our own city over 50 per cent of the children had had their tonsils removed some five years ago. It has always been incomprehensible to me how in the course of about ten years tonsillectomy became the most common pediatric procedure next to smallpox vaccination. In the city of London at one time 3 to 5 per cent of the children had tonsillectomies. A communication recently published stated that in some districts in London 50 per cent of the children had had their tonsils removed. In the city of Munich less than 10 per cent of the entire school population have had their tonsils removed, and their health record, compared with the health record in London, Paris, New York, and Rochester, is just as good. These results encourage a skeptical attitude toward tonsillectomy. The problem for us to consider is whether the tonsil is a factor in the vital economy of the child; whether it has a function essential to the welfare of the child; whether the child whose tonsils have been removed suffers as a result of the operation; whether the child whose tonsils have been removed has a better chance to escape infection than the child whose have not; and then perhaps also to give some consideration to the question whether the tonsils actually are of any value to the growing child.

Is tonsillectomy harmless? Postoperative mortality from tonsillectomy varied from 185 to 95 each year since 1922. That is not very many deaths for four or five hundred thousand tonsillectomies a year. The number of children who die of hemorrhage is difficult to learn because they are not generally listed as such, but, according to figures on small groups, there are a good many children in the course of a

year who die following operations because of immediate hemorrhage. When the number of children who die from lung abscess that might be directly attributed to the operation and, perhaps a less definite cause of death, pneumonia, meningitis, and perhaps the number of things that might be attributed to the operation are included, we cannot say without reserve that the operation is harmless.

There is increasing evidence to support the opinion that the lymphoid tissue plays some protective part in the mechanism of the young child and that these little children who have their tonsils removed for no good reason, except prophylaxis, will perhaps suffer from some other infection which might be more severe than an attack of tonsillitis.

Again I think it is very important to remember that you cannot compare a ten-year-old child with a five-year-old child. We all know that the incidence of infection at five years for certain diseases is very much higher than the incidence of different infections at ten years. In our control studies, we have compared children of the same ages, children who have had very much the same social environment and the same general type of food. It is likewise difficult to get suitable controls.

Mere physical inspection is insufficient to determine whether the tonsils are infected; nor is the size of the tonsil a satisfactory criterion. The pediatrician, the internist, or the practitioner is in a better position to decide indications for tonsillectomy. The course of the child's physical condition is probably the best index for determining whether a child's tonsils ought to be removed.

We are now testing the children who come up for tonsillectomy with a nucleoprotein of the hemolytic streptococcus, to determine if those who have a given allergic response to the hemolytic streptococcus are especially suitable for tonsillectomy. About 50 per cent of those tested reacted to the hemolytic streptococcic protein. After six months a certain number of these children lost their allergic response, a fact which is perhaps merely suggestive that many of these children had infected tonsils and that the others had large tonsils which had not become infected.

There are two outstanding indications for tonsillectomy. One is hypertrophy of the lymphoid tissue, including adenoids and tonsils, sufficient to give mechanical symptoms of obstruction. Although tonsils and adenoids may be hypertrophied in the winter, they may shrink to normal size in the summer. During the course of school examinations, it has been found that many tonsils spontaneously shrink in size, while being observed over a period of years.

The most common symptomatic complaint is interference with normal breathing. Tonsillectomy here gives excellent results. Good results have also been obtained in patients with dysphagia and halitosis. Speech defects or stuttering have been unmodified by tonsillectomy.

The second indication is infection of the tonsil. The tonsillar tissue takes care of itself in a great many instances. In studying these records it was found that the tonsillectomized children as a class are as well as the children who were not operated upon. Actually, their benefit has been greater than statistics prove because they probably represent the susceptible individuals, or they would not have been operated upon and would have been much worse had they not been operated on.

What are the indications in cases in which the tonsils are infected? What can be expected from operation in these children?

Bacteriologic study of tonsils indicates that the type of infection does not seem to have any particular bearing on the appearance of the tonsil.

Tonsillitis is still an important indication for tonsillectomy. However, removal does not guarantee against future infection, and it is a rather disappointing experience to have parents come back and say, "My child has a sore throat." Fifty per

cent of the children who were subject to two or more attacks of tonsillitis a year have been definitely relieved by tonsillectomy, not for one year but over a period of ten years. If a child has repeated attacks of sore throat after tonsillectomy, there is another factor to blame.

In the question of the rheumatic syndrome, the tonsil is very important. Tonsillitis is the most important preceding infection in rheumatism and occurs in a large number of cases of cardiac disease.

Tonsillectomy is not of any great value in the prevention of ear infections. The removal of adenoids probably is beneficial to younger children, but ear infections are seldom benefited to any great degree. Incidentally, more mastoid operations are reported in tonsillectomized children than in those with tonsils.

Cervical lymphadenopathy is a good indication for tonsillectomy. Tuberculous glands of the neck are one of the strongest indications for tonsillectomy. If there is associated pulmonary infection, tonsillectomy is useless. In a case of an active pulmonary tuberculosis in which there is tuberculous adenitis and the tonsils might be implicated, tonsillectomy may be done.

In sinus disturbances, postnatal discharges, and persistent headache, tonsillectomy has been disappointing. We find as much or more sinus disease in tonsillectomized children than we do in those with tonsils.

Statistical studies indicate that such pulmonary conditions as bronchitis, pneumonia, asthma, and tuberculosis have not been particularly benefited by tonsillectomy. In fact, there was a higher incidence of laryngitis, bronchitis, and pneumonia in tonsillectomized children.

Dr. Ruby Cunningham, at the University of California, in studying the incidence of pulmonary infections, found the same thing to be true, that there were more pulmonary infections in the tonsillectomized students than in those with tonsils. It may be that the tonsillectomized group represents the susceptible group.

The Rheumatic Infections.—Tonsillitis plays an important part in the causation of rheumatic disease. Tonsillectomy in children subject to tonsillitis distinctly aids in preventing rheumatic infection as shown by a study of disease incidence. From 10 to 30 per cent fewer children developed rheumatic infection in the tonsillectomized group. Likewise, the death rate from rheumatic disease was somewhat less in tonsillectomized children than in the others. As far as the question of recurrence was concerned, there was absolutely no difference. Removing the tonsils after the first attack did not influence incidence of recurrence.

Infectious Diseases of Childhood.—Tonsillectomy exerted no particular influence on the incidence of *scarlet fever.*

It is reasonable to expect a beneficial effect from tonsillectomy in preventing *diphtheria* because diphtheria is so often a tonsillar infection. Of course, there is a definite indication for tonsillectomy in curing diphtheria carriers.

Measles and *pertussis* were not appreciably benefited. In *head colds* there was possibly only a slight benefit. If colds are a virus disease, it is perhaps not reasonable to expect that taking out the tonsils would be of any great benefit in the prevention of colds. Children who have nasal obstruction are probably benefited by adenoid removal.

Renal Conditions.—There was slight benefit from tonsillectomy in patients with enuresis, recurrent pyelitis, and nephritis. Tonsillectomy after an acute attack of nephritis is rather dangerous. The operation should be done some time after the acute attack.

Nutritional Disturbances.—From statistical studies one cannot claim that the nutrition of individuals is very much improved by tonsillectomy although individual cases may show striking improvement.

Cyclic vomiting may be improved by tonsillectomy.

Asthma was not particularly affected by the presence or absence of tonsils.

The Mental Disturbances.—Some types of mental retardation due to respiratory obstruction or infection may be helped by tonsillectomy. Mentally defective and behavior problem children are not influenced by tonsillectomy.

That in a general way covers the symptoms as we encounter them in our practice. It is difficult to draw general conclusions as it is necessary to study the individual patient, but from statistics we might be able to surmise what can be expected in the individual case. When performed for proper reasons and definite indications, tonsillectomy is a very beneficial procedure.

It is important that these children be examined thoroughly from a general standpoint by the pediatrician, before any final judgment is passed by an otolaryngologist.

Bacteriology and Immunology of the Tonsils

DR. HORTON CASPARIS (NASHVILLE, TENN.).—The tonsils are exposed lymph tissue with a fairly large amount of surface which has a good many crypts. The tonsils probably play some part in building up immunity against common infections in the respiratory and gastrointestinal tract. A few days after the child is born, positive bacterial cultures can be obtained from the tonsils. Cultures of hemolytic streptococci vary from 10 or 15 to as high as 90 per cent. Other pathogenic bacteria found are pneumococcus, diphtheria bacilli, *Micrococcus catarrhalis*, influenza bacilli, and tubercle bacilli. Likewise, there is syphilitic and mycotic tonsillar infection, and more rarely, herpes virus infection. These infections may induce some degree of immune reaction in the tonsil. Repeated attacks have been known to become progressively milder, indicating a developing immunity.

There are some physicians who say that the tonsils function no longer after puberty and begin to atrophy. There are many who feel that it is quite necessary to keep the tonsils, at least for the first five or six years, simply because they are immunity-building organs and the severity of these infections is lessened when they occur later. The pathogenic significance of the hemolytic streptococcus is still in doubt. It is necessary to estimate the individual case on its own merits.

Tonsils and Tonsillar Infections

DR. I. A. ABT (CHICAGO).—Before proceeding with that portion of this review on the tonsil question allotted to me, I desire to refer briefly to what is known about the function of the tonsils.

The participation of the tonsils in the formation of new lymphocytes is the only firmly established function that can be ascribed to them. The structure of the tonsils is characterized by the infiltration of their epithelium with lymphocytes. A similar infiltration occurs along the entire digestive tract. It may occur diffusely, as in the small intestine, or in specific organs of the mucous membrane, as in the tonsils, the pharynx, or the solitary follicles and Peyer's patches in the intestine. It has been held that these collections of lymphocytes constitute a defense mechanism against the entrance of toxic products and microorganisms into the body. On the other hand, pathogenic bacteria may be found in the lymphoid tissue of the tonsils and follicles of the intestine in perfectly normal individuals. It has been suggested that the bacteria which gain access into the lymphoid tissue become modified and rendered less virulent, and thus come to act as vaccines, stimulating the production of antibodies, though it is recalled that the tonsils, as well as the follicles of the intestine, have been proved to be the atria of infection by different forms of bacteria and that general sepsis has resulted.

It has also been suggested that the tonsils may be glands of internal secretion. However, the proof for this assumption is entirely lacking. It has been assumed that because the construction of the tonsils is similar to that of the thymus gland and, that, since both are considered lymphoepithelial organs, both structures have similar functions, though evidence is lacking that the thymus gland itself functions as an organ of internal secretion in man. Certainly, extirpation of the tonsils has not shown any loss of recognizable endocrine function.

Tonsils as Foci of General Infection.—It is a generally accepted fact and hardly needs repetition that the tonsils may be the foci for general infections, producing metastasis in various parts of the body. Clinical observations have frequently shown that nephritis, carditis or endocarditis, meningitis, and rheumatism, as well as general sepsis, bear a causal relation to tonsillar infections. Such infection may be acute or chronic. In the acute infection there may be an associated pharyngitis. The tonsils become swollen, the excretory ducts of the crypts and the lacuna become closed, and eventually the crypts are filled to bursting. The microorganisms are now enclosed in this stagnating mass which offers an excellent culture material. The plugs which close the crypts of the tonsils may be extruded and drainage may occur though, on the other hand, toxic products which have been formed in the crypts may burrow deeper through the loosened epithelium and gain access to the body through the lymph stream.

Though following acute tonsillitis the little patient frequently makes a perfect recovery, in some cases the tonsils may remain enlarged after an acute attack, resulting in a so-called hyperplasia, which is characterized by the persistence of crypts containing infected material and closure of the excretory ducts. This condition predisposes the patient to recurrent attacks of angina, which present themselves as acute tonsillitis. A chronic inflammatory condition of the tonsils may persist in large as well as in small tonsils, and these persistent foci may produce general infection through the lymph tracts, or they may be the source of protracted ill health.

In adults, after the tonsillar tissues have undergone involution and have been replaced by connective tissue, the crypts may still persist and serve as a breeding place for bacteria and become foci for systemic infections.

The question of deciding whether tonsils are actually the portals of infection or the primary foci is discussed in every treatise on this subject. An important factor is a positive history of infection; however, the size of the tonsils should not be taken as a criterion, for a small tonsil may be the site of serious infections. Superficial redness or dilated blood vessels suggest disease of the tonsils. Bacteriologic smears or cultures made from the tonsils need have no definite etiologic significance.

Although it is sometimes easy to determine that a secondary infection has resulted from tonsillitis, at other times the decision is difficult or impossible. It has been maintained that there is a relationship between angina and appendicitis, also that certain skin diseases such as purpura and erythema multiforme may follow acute infection of the tonsils. It may be accepted as a general proposition that general sepsis arises from special foci of tonsillar or peritonsillar infection. As such are to be considered peritonsillar and retropharyngeal inflammations and abscesses, or thrombophlebitis originating in the tonsillar veins and continuing its course into the jugular vein. More recently, suppuration in the parapharyngeal space has been emphasized as a source of postanginal sepsis. This space is closely connected by blood and lymph vessels with the tonsils, and infection of the tonsils and peritonsillar tissues has ready access to the parapharyngeal space.

While a general systemic infection may occur during an attack of angina or peritonsillar abscess, it must be remembered that these remote infections may develop when an acute tonsillitis is subsiding or days or weeks thereafter.

At this point a brief anatomic description of the retropharyngeal and parapharyngeal spaces should clarify our clinical conceptions.

The retropharyngeal space lies anterior to the vertebral column and extends upward to the base of the skull and downward toward the posterior mediastinum. It lies between the pharynx in front and the vertebral column behind. It is enclosed anteriorly and posteriorly by two layers of fascia. It invests the superior constrictor of the pharynx and is continued forward on the buccinator muscle. Laterally it blends with the sheaths of the great vessels. There is a chain of lymph nodes on either side of the midline between the pharyngeal and prevertebral muscles.

The parapharyngeal space is the region of connective tissue which is separated medially from the tonsil by the constrictor muscles of the pharynx and the parapharyngeal fascia. The lateral boundary is formed by the internal pterygoid muscle, the ascending ramus of the lower jaw, and the capsule of the parotid. The space extends upward to the base of the skull and downward along the esophagus and larynx into the posterior mediastinum. It is filled with connective tissue and fat, as well as important vessels and nerves. Here are found the carotid, the internal jugular, the ninth to twelfth cranial nerves, and the sympathetic nerves. There is also a rich network of small veins and numerous lymphatic glands.

An opening in the parotid fascia communicates this space with the parotid gland. Through an opening at the styloid process infection may spread from the pharyngeal space into the parotid gland, and from here, upward to the bony and cartilaginous ear, which may become actively involved.

Peritonsillar Abscess.—While in adults one of the most frequent local complications of tonsillitis is the so-called peritonsillar abscess, also known as phlegmonous tonsillitis and quinsy, this complication is, however, uncommon in infancy and early childhood. Not only the peritonsillar tissue, but the tonsil itself, is often involved. Essentially, however, the suppuration occurs in the loose connective tissue behind the tonsillar capsules. The abscess may arise about the paralacunar abscesses which spread to the capsules of the tonsils, eventually rupture through the latter, and invade the loose peritonsillar connective tissue. The peritonsillar abscesses usually develop when the angina is subsiding or has recently subsided.

The peritonsillar abscess is characterized by fever and pain and difficulty in swallowing; it is usually unilateral, though it may involve both sides. On examination, one observes that the tonsillar region is red and swollen, that the palate is usually arched forward, and that the tonsil is pushed toward the middle line. Frequently the abscess ruptures spontaneously.

The diagnosis is usually suggested by the appearance of the patient. He has difficulty of articulation, his speech is altered as though he had a foreign body in the pharynx; he holds his head to one side and has difficulty in opening his mouth. Occasionally, no pus is found even after incision has been made. This condition is undoubtedly due to simple inflammatory edema. The best results in evacuating the pus are obtained if incision is delayed until definite fluctuation of the abscess is elicited.

The complications of peritonsillar abscess are similar to those of acute tonsillitis. Among these may be mentioned hemorrhage, laryngeal edema, and aspiration of pus into the respiratory tract. Those abscesses which lie posterior to the tonsils may cause a collateral edema of the lingual surface of the epiglottis, or the edema may spread to the arytenoepiglottic folds and into the sinus pyriformis. Displacement of the larynx and obstruction of its lumen may lead to stenosis, and not infrequently to a fatal termination. Hemorrhage may occur as a result of the erosion of important vessels by the infiltrating abscess, or there may be bleeding following the incision of the suppurating focus, due to the injury of a blood vessel. As a rule, this bleeding can be controlled by local methods.

Severe hemorrhage may occur after peritonsillar or retropharyngeal abscess complicated by suppuration in the parapharyngeal space, as well as in cases of severe cervical abscess or cellulitis. In the patients who have been studied it has been found that the common carotid, the internal carotid, less frequently the external carotid, the internal jugular vein, the superior laryngeal artery, the lingual artery, and the pharyngeal artery are the vessels most frequently involved. Unsuspected aneurysmal dilatations of the common carotid and its branches may lead to hemorrhage during various operations in the pharynx and on the tonsils, as well as during pharyngeal and parapharyngeal suppurations.

The citation of a case which occurred in our own practice will illustrate the onset and course of a hemorrhage following a pharyngeal abscess. N. C., an eight-year-old boy, fell ill during the winter of 1933 with severe tonsillitis followed by a diffuse phlegmonous process in his pharynx. He had considerable fever, difficulty in swallowing, earache, but no otitis. The pharyngeal abscess was not incised. After he had been ill for about two weeks, we were summoned because he had suddenly developed a severe spontaneous hemorrhage of the throat. The blood was gushing from his mouth and through his nostrils. All local attempts to stop the hemorrhage or to find the bleeding point were unsuccessful. The boy was immediately removed to the hospital where the otolaryngologist and a general surgeon proceeded to ligate the internal carotid artery on the left side, the point from which the hemorrhage seemed to come. He seemed better for twenty-four or thirty-six hours, after which he had another profuse hemorrhage; he became faint and pallid and showed evidence of shock; a transfusion was performed. In twenty-four hours he had another severe hemorrhage which nearly exsanguinated him. At this time it was decided to ligate the common carotid on the left side. This was done as rapidly as possible, and after the ligation the patient received another transfusion of about 300 c.c. of blood. He was returned to bed, and the hemorrhage ceased. No further hemorrhages occurred; the boy made a complete recovery and is perfectly strong and well.

A retropharyngeal abscess is an inflammation of the pharyngeal lymph nodes with secondary cellulitis and suppuration. It occurs most commonly in infancy and young childhood. It has been maintained that the disease never occurs when the tonsils have been removed, but this is probably not correct. The abscess is not necessarily preceded by tonsillar inflammation. It occurs rarely during scarlet fever or measles, and it may complicate a severe erysipelas of the pharynx. The retropharyngeal abscess may be due to an injury of the pharyngeal wall caused by swallowing hard-pointed substances, or it may arise from suppuration of the lymph nodes or nasal pharyngeal infection of any kind. In rare cases the abscess may be caused by tuberculous cervical spondylitis. Retropharyngeal abscess may occur after tonsillar enucleation and may lead to infection and suppuration of the retropharyngeal lymph nodes. An abscess of considerable size may develop.

Abscesses of the Tonsils.—Single or multiple abscesses may occur in the tonsillar tissue. They may lie superficially and arise in the crypts of the tonsils, or they may be located centrally and lead to inflammation and swelling of the tonsils. Not infrequently they rupture spontaneously.

Vincent's Angina.—For the purpose of completeness a brief reference is made to this condition. It occurs in persons of all ages. The cause of the disease appears to be a symbiosis of the fusiform bacillus and the spirochete. It occurs most frequently in debilitated persons. The organisms may be found in healthy subjects in the crypts of the tonsils and on unclean teeth. The condition may be confused with diphtheria and syphilis, from which it may be readily differentiated by closer examination. On examining the mouth and pharynx, one frequently finds an exudate under which lies an ulcer, or in the milder cases, the exudate may be removed, leaving a bleeding surface. The cervical lymph nodes may be enlarged. They rarely sup-

purate. Severe types are observed with grave general symptoms. Though in the milder cases improvement occurs in a few days to several weeks, in the severer types, ulcerative stomatitis, necrosis of the tonsils or jaw, and otitis media may occur.

Agranulocytic Angina.—Agranulocytic angina, also called granulopenia or the neutropenic state, has been described by a number of authors. The disease is characterized by reduction in the number of leucocytes and by the disappearance of the granulocytes. I do not intend to give an exhaustive description of agranulocytic angina, but to trace its connection, if there is any, to preceding tonsillar disease. In the series of cases reported by Givan and Shapiro,* there is a review of about twenty-nine cases. With only four exceptions, all of them had definite antecedent history of acute infections of one kind or another, leading to the onset of agranulocytosis. The preceding disease varied. Some of the patients suffered from gingivitis, tonsillitis, otitis, tooth extraction; others had pyodermia, osteomyelitis, and acute upper respiratory infections.

It has been held that the necrotic, ulcerated, membranous lesions occurring on the tonsils and in other regions are secondary to the granulopenia. It has been thought that the white cell count is low before the necrotic lesions develop.

The differentiation of agranulocytic angina and tonsillar general sepsis is possible only by the examination of the blood. The cause of agranulocytic angina is not known although several theories have been suggested.

The treatment is for the most part unavailing. Blood transfusions give only temporary relief, if any, use of roentgen rays is of no proved value. A preparation of nucleotide may stimulate the bone marrow, and it is thought to be of value in cases of primary granulopenia.

Monocytic Angina.—Another form of necrotic angina is the so-called monocytic angina. The tonsils are large and inflamed, they are covered with a purulent, greenish, membranous exudate, with an underlying superficial necrosis. The affection is often unilateral. The patient usually suffers from elevation of temperature, with the constitutional symptoms associated with a general infection. The lymph nodes are swollen; the spleen is also frequently enlarged; the liver, more rarely. The diagnosis is established from the blood examination. The total leucocyte count is moderately or relatively high, though there is a marked increase in the mononuclear cells. The course of the disease is nearly always favorable. The principal point to be emphasized is the value of the blood examination. One can at once differentiate an agranulocytic angina with its characteristic blood findings from a monocytic angina characterized by leucocytosis and mononucleosis.

Tuberculosis of the Tonsils.—Tuberculosis of the tonsils has often been observed, especially in those cases in which the condition has been sought. Generally one finds miliary nodules, less frequently, tuberculous ulcers. Sternberg reports the case of a child eight years old who died of general tuberculosis and who showed tuberculous ulcers on the tonsils. In thirty-two autopsies on infants, Geipel found thirteen cases of tuberculosis of the tonsils, though these may not have constituted the primary lesions of the infection. In general it may be said that the tonsils are not frequently the atria for tuberculous infections, and primary tuberculous lesions in the tonsils are extremely rare.

In this short sketch I have very briefly mentioned some of the complications of the diseases of the pharynx and tonsils, and in order to avoid repetition I have omitted to refer to the cardiac, renal, nervous, and other complications, which have been assigned to the other participants in this symposium.

*Givan, T. B., and Shapiro, B:. Am. J. Dis. Child. 46: 550, 1933.

DISCUSSION

DR. SAM P. WAINWRIGHT (BIRMINGHAM, ALA.).—Dr. Kaiser mentioned certain accidents that occurred during a tonsil operation, such as deaths from anesthesia, which may be of an allergic nature, and also postoperative hemorrhages. I suggest the importance of preoperative treatment in prevention of hemorrhage and unpleasant allergic reactions.

I routinely give patients for tonsillectomy a high protein diet along with viosterol and calcium and have had a minimum of hemorrhagic complications.

DR. KAISER.—We have given no calcium or viosterol to the large number of patients—over twenty thousand now—who have been operated on in the clinic. The great majority, however, have had cod liver oil or viosterol, or both, prior to their operation. Most of the otolaryngologists give calcium for a number of days prior to the operation and then test the blood coagulation time.

The other question which might be brought up in connection with preoperative treatment is one concerning the thymus gland. In Boston it is felt that no tonsillectomy should be made without an x-ray examination of the thymus gland. We have operated upon twenty thousand children in the clinic, and there have been six deaths immediately associated with the operation. All six of these children were autopsied; but none of them had any thymus disturbance.

All cases are hospitalized the night before, are examined, and are refused operation if there is fever.

DR. A. G. MITCHELL (CINCINNATI).—It has already been mentioned and very well stated by Dr. Abt that we really do not know very much about the function of the tonsils except as part of the lymphoid structures in the body. Certainly there is no evidence that removal of the tonsil is particularly detrimental.

It is interesting to note that one never removes from the nose and throat all the lymphoid structures present. After tonsillectomy there is hypertrophy of the remaining lymphoid structures. Is this remaining tissue with its hypertrophy potentially a source of difficulty from the point of view of general distribution of infection?

With regard to the bacteriology of the nose and throat, there is still much to learn. In tonsillectomized persons there are the same type of organisms, perhaps less in number.

Coburn has reported his experience with children who had rheumatic infections and were hemolytic streptococcus carriers. They were removed to Porto Rico where it is said that hemolytic streptococci are very seldom, if ever, present in the noses and throats of the people. These children during their sojourn had no recurrence of rheumatic manifestations. Some of them were returned to their previous environment with the return of hemolytic streptococci and recurrence of their rheumatic manifestations.

With regard to age incidence, May Wilson has reported that rheumatic conditions continue from approximately four years of age, increasing up to nine or thereabouts, and then the curve of incidence decreases after puberty.

Repeated respiratory infections are very common in early childhood.

I think Dr. Abt mentioned the matter of peritonsillar infection and the statement that is often made that these occur only when the tonsils are present. That is not so because they do occur when the tonsils are not present. It may occur when the tonsils have not been properly removed.

Detrimental effects after tonsillectomy should not be overlooked. One of these is increased incidence of lower respiratory tract infections.

Statistical studies based on parental information are unreliable. The merits of tonsillectomy must be decided by the status of the individual patient.

DR. JOHN D. STEVENSON (BEAVER, PA.).—Has any particular study been made of the effect of climate upon tonsillitis?

DR. KAISER.—As Dr. Mitchell has indicated, in the Coburn studies, in warmer climates there are fewer streptococcic infections and fewer cases of tonsillitis. In our extreme southern Gulf Coast regions and in the West Indian Islands, there are fewer of those infections. In Canada and England, there is a much higher incidence of streptococcus infections and rheumatic fever. In Arizona and in high dry climates there is likely to be fewer respiratory infections.

Five years ago I was in Africa and went from Cairo up the Nile to central Africa. All the way I examined the tonsils of children wherever I had a chance and was amazed to find that the farther inland I went the smaller the tonsils became. Of course, these were all untreated natives of wild tribes. In the southern Sudan and in the Belgian Congo where I spent some time, I could not find tonsils in a large number of those children. The medical missionaries and government officials whom I met said the natives never had any ear trouble, tonsillitis, or swollen glands unless caused by sleeping sickness. Climate must have something to do with it. Whether the hypertrophy of the tonsils, as we see it in the East, is a result of the infection or Nature's efforts to guard against the infection, I do not know.

DR. JAMES DUNN (DAVENPORT, IOWA).—How many attacks of tonsillitis should a child have had before tonsillectomy is considered?

Personally, I have always felt that during an acute attack the tonsils should not be removed, but we have members of the profession in our community who say that since we remove the appendix when it is acutely infected, why not remove the tonsil when it is acutely infected.

DR. KAISER.—I will answer the second question first. I absolutely disagree that the tonsils should be removed when acutely inflamed. The age of the child and the extent of recovery are the chief criteria in deciding whether tonsillectomy is indicated after repeated tonsillitis. Thus, a child of two or three years who recovers completely from attacks of tonsillitis needs no tonsillectomy.

DR. LOUIS H. SEGAR (INDIANAPOLIS).—There may be exceptions to this age limit. I had under my care an infant with repeated attacks of tonsillitis from the third to sixth month of life. When the infant was nine months old, a tonsillectomy was performed, after which the infant was perfectly well.

As pediatricians, we must impress the otorhinolaryngologists as to the importance of a thorough general examination of the patient from a pediatric standpoint before tonsillectomy is done.

DR. HERBERT E. HALL (UNIONTOWN, PA.).—Enough statistics have become available for a guide as to the general conception of the indications for tonsillectomy. Personally, I have seen more first attacks of rheumatic carditis, rheumatic fever, and chorea in patients who have had their tonsils removed than in those who have not.

DR. W. C. FARGO (CLEVELAND, OHIO).—The remarks I wish to make are prompted largely by an experience of about thirteen years in connection with the Children's Fresh Air Camp here in Cleveland where I have under my supervision daily from sixty to a hundred youngsters. Some of these children progressed poorly from early infancy. They had poor feeding histories and weighed 16 or 18 pounds at the end of the first year. These continue below normal, regardless of tonsillectomy.

As regards postoperative care, it is desirable that the children be kept in bed until the throat is healed. I have had the opportunity of observing the throats in tonsillectomized children daily over a period after the tonsils had been removed. The exudate never disappears under ten days, and it is usually fourteen days before the tonsil sites are completely epithelized. The sites from which the adenoids have been removed also heal slowly.

Some of these throats become very acutely inflamed, and the uvula frequently reaches a size six or eight times its normal size. These children run a temperature; the cervical glands are enlarged; and it is exceedingly difficult to swallow. Observation of weight records show that children lose from 3 to 8 pounds after a tonsillectomy.

DR. WILLIAM WESTON (COLUMBIA, S. C.).—I was particularly impressed with the tremendous reaction voiced against the indiscriminate removal of tonsils when this subject was discussed at the last meeting of the American Laryngological Association. Many otolaryngologists are treating these patients through dietary measures.

DR. J. A. JOHNSTON (DETROIT, MICH.).—What is the opportune time to perform a tonsillectomy after acute fever? I have seen a number of recurrences which I felt were due to too early operation.

DR. JOHN F. CAREY (JOLIET, ILL.).—I find that the early removal of tonsils does tend to increase hyperplasial lymphoid tissue in the pharynx. I practice in an industrial community of about fifty thousand population where recently we had a preschool roundup. Sixty-five per cent of those children had had their tonsils removed, and 95 per cent of the remainder were advised to have them removed.

I feel definitely that tonsillectomy has not lessened the attacks of streptococcic infections that develop in the pharynx. Abdominal pain is frequently noted in children when they have this hyperplasia in the pharynx. Likewise, long-continued fever may be due to this lymphoid hyperplasia.

DR. ARTHUR ABT (CHICAGO).—I had the opportunity of observing over one hundred children with rheumatic heart disease who were attending a special school. When these children are admitted to this school, the question is always asked in the history, "Did the rheumatic disease develop before or after the tonsils were removed?" In many of these children the first sign of rheumatism, joint rheumatism followed by the cardiac condition, developed after tonsillectomy.

I agree with Dr. Graeme Mitchell as to the importance of age incidence. I believe that peritonsillar or retropharyngeal abscesses are more common in very young babies.

The case of the pharyngeal abscess with arterial bleeding mentioned by Dr. Isaac Abt was in a boy eight years old. In the last year we have seen in our practice four girls, four years or older, whose tonsils had been removed, with parapharyngeal abscess and cervical adenitis. It was very interesting to discover that children over four years of age who had had their tonsils removed developed this parapharyngeal infection.

When would you rather operate; in winter or at the end of summer? Perhaps in summer the tonsils would be in better condition for removal than during the late winter or early spring.

CHAIRMAN ABT.—In Spokane there has been a considerable number of cases of poliomyelitis lately. The physicians feel that they must not perform any more tonsillectomies during this time because they find that most of the poliomyelitis attacks occur in tonsillectomized children.

DR. EDWIN CLAY MITCHELL (MEMPHIS, TENN.).—Does a high carbohydrate diet tend to hypertrophy the lymphoid tissue in this region?

As a rule, only the infected tonsil is removed. Do you find it necessary or beneficial in certain conditions to remove the lingual tonsil later or to drain the sinuses?

DR. O. W. ROWE (DULUTH, MINN.).—I have never seen a peritonsillar abscess or glandular disease in a child who has had a complete tonsillectomy. As regards cyclic vomiting, Dr. Sedgwick twenty years ago recommended tonsillectomy. We have been following his advice with results in more than 50 per cent of the cases. I have seen migraine and epilepsy develop in children who have been cured of cyclic vomiting by tonsillectomy.

DR. H. B. HAMILTON (OMAHA).—With reference to Dr. Rowe's statement that he had never seen parapharyngeal abscess in a tonsillectomized child, I may say that I have one under observation at the present time.

I am convinced that coal tar products are abused in the treatment of the throat infections. There is a demand on the part of the people that the fever be controlled to the extent that I think many men are tempted into the overuse of the coal tar products.

I am quite convinced that a lot of our beneficial results are the result of adenoidectomy rather than tonsillectomy.

DR. ELI FRIEDMAN (BOSTON, MASS.).—Dr. Kaiser said there was no relation of asthma to the tonsils. This is a debatable question. I had a child who developed asthma after tonsillectomy.

Scarlet fever seems to be more severe in patients who still have their tonsils.

DR. H. H. PERLMAN (PHILADELPHIA).—I would like to ask if the essayist has histologic studies of removed tonsils. It seems to me that in this enthusiasm for wholesale enucleation of tonsils, perhaps more normal tonsils are removed than should be.

The second question I would like to ask is whether the local use of various dyes in gargling or swabbing the throat has any effect in curtailing so-called acute tonsilitis? I believe it is also timely to speak of the use of drugs which have been exposed as a cause of agranulocytosis, particularly pyramidon.

DR. KAISER.—It is of great importance, as Dr. Graeme Mitchell brought out, to know about the age incidence of tonsillitis in children. Tonsillitis has an incidence of about 10 per cent in children of preschool age; about 25 per cent in children between five and ten years of age; and then in later years drops off, so the incidence in a given community of these infections must be known to compare controls with so-called operative children. We have tried to do that in some of our studies. The age incidence has been particularly stressed in connection with rheumatic disease, and the benefit of tonsillectomy has been noted in the so-called peak age, i.e., between five and ten years of age. After ten years of age no benefit could be seen in either primary attacks or in the later course of the disease. A similar analysis should be made of each one of these complaints.

Postoperative care of tonsillectomy, as Dr. Fargo has brought out, is often neglected and should be given much more attention. Dr. Shick suggests that tonsillectomy disturbs the entire metabolism of the child for a time. Insist upon five days in bed after a tonsillectomy, no matter what the condition may be, and much longer after severe illness. Too much food will cause vomiting and bring on certain complications.

The dietary factor is of great interest, and some day it may play a very important part in our conception of this question. We became interested a short time ago

in the relationship of vitamins to rheumatic disease. It has been found that animals fed on a diet deficient in vitamin C developed scurvy and symptoms which simulated rheumatic infection. In testing the effect of vitamin C on rheumatic patients, we found eight times as much vitamin C in the tonsillar tissue as in the blood. But there does not seem to be a deficiency of vitamin C in the diet of these children who have rheumatic infections. Vitamin A could not be found in any of the tonsil tissue.

The optimal period for tonsillectomy after an attack of rheumatic fever is a matter of individual opinion. Tonsillectomy rarely prevents recurrences, and if the operation is performed immediately after an acute attack, an endocarditis may start up. Chorea also occurs frequently after tonsillectomy. For that reason every patient should have a month or six weeks elapse before the operation. There should be a normal white count and the pulse rate under a hundred per minute before one advocates tonsillectomy. The temperature does not make so much difference as the pulse and sedimentation rates.

A few years ago Syracuse had three or four times as many cases of poliomyelitis as Rochester. The health officer felt it was because there were fewer tonsillectomized children in Rochester. The situation was reversed later, and Rochester had three times as many cases as Syracuse. Tonsillectomy gives absolutely no protection against poliomyelitis. It is unwise to operate during an epidemic period of virus disease.

I doubt very much whether pyuria is cured by tonsillectomy. In few instances, in which the pyuria was a result of chronic infection in the tonsil, tonsillectomy may relieve the pyuria. It may be tried if every other procedure has failed.

In a study of children with asthma, removing the tonsils seemed to make no difference. After tonsillectomy some children have reacted to an infection that was localized in the tonsil.

The same thing is true of scarlet fever. There is a very slight advantage for the tonsillectomized child. The period in the hospital is a day or two shorter; there are fewer streptococci in the throat and fewer complications, but the benefit is so small as to be of hardly any value.

On the basis of some experimental work, I believe that not only the tonsillar tissue, but all lymphoid tissue in the nose and throat, plays a part in local immunity. It may be vitamin content; it may be an enzyme; and it may be internal secretion which plays some part in protecting individuals against infection.

DR. CASPARIS.—The one encouraging thing is that we are giving a great deal of thought to the individual who has tonsils. We are accumulating information from time to time which helps to decide when tonsils should or should not be removed.

Tonsils are very valuable organs, and the time to remove them is when they become more of a handicap than a benefit to the individual. We must study our patients as individuals. We must take into consideration the appearance of the tonsils—whether or not the crypts are closed, and the presence of dilated blood vessels over the anterior folds of the tonsils.

I would like to make one point in this connection. A number of children are being examined superficially by school physicians. We have asked the State, County, and City Health Departments to formulate this policy: that a school physician should never say that a child ought to have his tonsils removed but should say to the parent, "See your physician about your child's tonsils," should advise that parent or child to see about this particular defect and should not give recommendations as to what should be done about it.

DR. JOHN L. MORSE (BOSTON).—I agree entirely with Dr. Kaiser that too much attention is paid to the tonsils and altogether too little to adenoids. A great

many infections attributed to tonsils are really infections of adenoids. Most of the obstructive symptoms attributed to tonsils are due to adenoids. Inflammation of tonsils alone is very seldom accompanied by ear complication. They are due to infection of the adenoids.

I advise removal of adenoids for repeated colds, for trouble with the ears, for difficulty in breathing, and also for difficulty in swallowing. I dislike very much to remove the tonsils in a child under five years of age although Dr. Kaiser said that under certain conditions age makes no difference.

The indications which I consider sufficient to justify tonsillectomy are tuberculosis of the glands in the neck, repeated attacks of tonsillitis, and cervical adenitis which persists after tonsillitis. However, I have observed children whose parents had neglected my suggestion of tonsillectomy for repeated tonsillitis and cervical adenitis. Several years later the tonsils appeared normal, and the indications had spontaneously disappeared.

After tonsillectomy there is hypertrophy of lymphoid tissue on the posterior pharyngeal walls. Tonsillectomy and adenoidectomy does not remove all the lymphoid tissue in these regions.

My general conclusion would be that adenoids should be removed oftener than at present and tonsils much less frequently.

DR. HERMAN SCHWARZ (NEW YORK CITY).—The evaluation of the benefits from tonsillectomy is difficult. I doubt the existence of a pure acute tonsillitis. There is always an inflammation of the other lymphoid tissues. On the other hand, I have to turn around and say that there is no question but that, as Dr. Kaiser has brought out so well, the age when tonsillectomy is done is important so far as immunity is concerned. We studied seventy-eight histories from this point of view. In those under five years the results were not very good. From five years up the results were good.

The indications for tonsillectomy given here today are very conservative, and I agree with them absolutely. It gives me a great deal of pleasure to note this attitude.

Also, I should like to discuss pharyngeal sepsis. It occurs especially in very young infants and has no relation to the tonsils. This sepsis is followed by streptococcemia. There may result metastatic abscesses; with surgical treatment recovery results, or peritonitis, or meningitis with a fatal result may develop. In another type of upper respiratory infection with streptococcemia there is otitis media and sinus thrombosis. Metastases occur in the lungs and joints. There are two types of pharyngeal sepsis; a benign type with recovery and the other with septicemia and a very high mortality.

Finally, a word about chorea. Most of these patients developed chorea long after the tonsils were removed.

DR. ALBERT J. BELL (CINCINNATI, OHIO).—As Dr. Morse has said, too little attention probably has been paid to the adenoids; in all probability the removal of adenoids might have done what the combined operation achieved. Unfortunately, the tonsils might become infected later and necessitate a second operation.

While it may be safe to say that by removing the tonsils the chief source of infection in the throat has been removed, they are only part of the lymphoid tissue in the throat. It is possible that infected tonsils may act as an allergic factor and even produce an immunity against the particular bacteria.

DR. KAISER.—It is important to be careful about the criteria and the time elapsed in comparing results after tonsillectomy. We should compare the effect on specific symptoms at least five years after tonsillectomy for any trustworthy

statistical information. I have used as many private cases as dispensary cases. If you compare them complaint for complaint, then the results are practically the same in private and in dispensary practice.

In some instances tonsillectomy has been advised for the prevention of children's diseases or for improving the general condition. Such vague indications tend to bring the operation into disrepute. I have become very cautious about advising the removal of tonsils and adenoids to increase the child's appetite, to improve his general condition, or to prevent colds.

DR. ALLAN L. RICHARDSON (DETROIT).—Dr. Canfield has described the dolichocephalic blond type in whom there is excessive regeneration of lymphoid tissue. Incidentally this type of child is particularly subject to anesthesia reactions on the operating table.

May I ask whether there are patients with an allergic background who have unfavorable results after a tonsillectomy even to the extent of developing asthma?

DR. CASPARIS.—There have been some cases of this nature, but I do not know the explanation.

CHAIRMAN ABT.—That was mentioned in the round table conference on allergy. Tonsillectomy has been the deciding factor in the allergic state of the child.

DR. J. V. GREENBAUM (CINCINNATI, OHIO).—What is the opinion about removing the tonsils only?

Based on the work of some English observers who put blue dyes in carious teeth and observed the dyes deposit in the tonsils, we made a rule in our hospital that no child should have his tonsils taken out if his teeth showed a number of cavities. The teeth should be in as perfect condition as possible before the tonsils are removed.

DR. KAISER.—I doubt whether the same results would be obtained if the adenoids were not removed.

Our clinic was started in the dental dispensary. The primary purpose was to clear up the teeth of these children. No child could have his tonsils and adenoids out until he had been through the dental dispensary and had all the infections cleared up. There was no difference in the two groups—those who had their infections cleared up and had their tonsils removed and those who had clean teeth and had their tonsils removed.

DR. WALTER B. STEWART (ATLANTIC CITY, N. J.).—Do the tonsils clear up after the badly infected teeth are removed?

DR. KAISER.—Badly infected teeth will often cause some of the symptoms that we ascribe to infected tonsils. We found that 20 per cent of the children who had cervical adenitis got it from infected teeth and not from infected tonsils. School examinations have failed to take into consideration that infected teeth should be cared for before the tonsils are removed.

DR. FRANK LEECH (WASHINGTON, D. C.).—Did the children with infected teeth have root abscesses or carious teeth?

DR. KAISER.—Most of them had infected gums.

DR. WARRIN R. SISSON (BOSTON).—Of all the indications for tonsillectomy, mechanical obstruction seems to be the only reliable one. It would be a great service if this organization should reduce the needless indications for tonsillectomy.

DR. CASPARIS.—Sometimes it takes a year to decide whether or not tonsils should be removed. It is necessary to study the history, the physical findings, and the general health of the child before deciding on tonsillectomy.

DR. FRANK T. MITCHELL (MEMPHIS, TENN.).—There is still a tendency to make the whole decision on the basis of one examination and not to give the thorough consideration that has been spoken of here. I think that explains why in a study made by the Metropolitan Life Insurance Company it was found that as many as 60 per cent of the eleven-year-old children have their tonsils out. It was also found that a large part of that influence was brought to bear by school nurses, and all they had to go on was a look at the tonsils made by the school physician instead of an examination based upon a thorough history and study of the individual. I think it is very important that the members of the Academy influence favorably this public health problem.

DR. STANLEY NICHOLS (ASBURY PARK, N. J.).—We should follow up cases from two to three years after tonsillectomy in order to record the end-results. We found x-ray treatment ineffective and followed by recurrences within six months to a year. In children in whom for one reason or another it is necessary to postpone operation, it is still worth while to try x-ray treatment.

School nurses have a habit of saying that the tonsils must come out if there is a moderate cervical adenitis present. I have had the experience of looking at large numbers of children in the spring and in the fall. In the fall there are about 30 per cent of those glands moderately palpable, and in the spring there will be about 90 per cent of them. The school nurse sometimes gets into difficulty here.

Finally, we should recommend that all children have a thorough pediatric examination before having the tonsils removed by an otolaryngologist.

CHAIRMAN ABT.—I am going to ask Dr. Morse to discuss the dentition aspect of this subject.

DR. MORSE.—I feel quite certain that I have seen more than one baby, just before a tooth erupted, whose gum was swollen and purple, who lost his appetite temporarily, was unusually fussy and perhaps had a little temperature. I think I can also remember two or three babies that at about that same stage would have loose bowels. Outside of that I don't think that I have ever seen any disturbances which I thought could be attributed to dentition.

DR. KAISER.—The increased incidence of lower respiratory infections occurred at all ages, but more particularly in the younger children. Perhaps you are familiar with Dr. Ruby Cunningham's figures based on a statistical study at the University of California. She found that the tonsillectomized students had a higher incidence of lower respiratory infection. She explained it on the ground that they were a susceptible group.

Personally, I have never felt that the teething was a factor in respiratory infection. I think it is a question of the prevailing infection in the community, rather than teething.

DR. CASPARIS.—The efficacy of vaccines in the treatment of "colds" is a difficult matter to evaluate. There is a possibility that colds are not infectious but allergic reactions.

CHAIRMAN ABT.—Dr. Barnett, will you tell us about your operative procedure in relation to the poliomyelitis?

DR. E. J. BARNETT (SPOKANE, WASH.).—There is a clinical experience in this field that I should like to report. We have had in our community annually in July and August what might be called an epidemic of infantile paralysis. It is rather peculiar that infantile paralysis in our community is usually the bulbar type, but the striking fact is that the majority of these cases of the bulbar type of infantile paralysis come on immediately after tonsillectomy. In the presence of this

annual epidemic at the same time each year, it may be pointed out that tonsillectomies are usually done before entrance into school. About the third or fourth day after tonsillectomy many of these children develop bulbar poliomyelitis. These children do not have a preparalytic stage. Those who develop infantile paralysis while they are convalescing from tonsillectomy never have the spinal type. The mortality in these children with bulbar poliomyelitis following tonsillectomy without preparalytic stage is terrifyingly high. For this reason when our annual epidemic begins, we insist that all operations be held up.

DR. THEODORE ELTERICH (PITTSBURGH, PA.).—A few years ago one of the physicians of Pittsburgh reported that a child who previously had had encephalitis precipitously developed a nervous syndrome soon after the anesthetic was given. A neurologist who was there said it was his experience that one must be very careful in giving an anesthetic to an individual who has had encephalitis. We have all had the experience that children who show mild symptoms of chorea develop a precipitious chorea when they are subjected to an anesthetic at that time. The thought occurs to me that it is not the operation but perhaps the anesthetic.

DR. KAISER.—We have studied some twenty thousand records of children who came in for operation; none of them had x-ray examinations of the thymus. Five or six died immediately after the operation; three died from hemorrhage, and in the others the cause was unknown. The autopsies showed normal thymuses as far as the weights were concerned. The children were not blonds. They happened to be Italians and all brunets.

The diminished incidence of rheumatic fever is probably true but must be correlated with the differences in various communities. I doubt very much if there is any marked difference in rheumatism or scarlet fever after tonsillectomy. One may see the lessened incidence in communities in which few have been tonsillectomized. I believe it is due to changes in severity of the disease rather than the effect of tonsillectomy.

Chorea certainly is a disease which is not benefited by tonsillectomy as far as the symptoms are concerned. There is a tendency in our figures to show that the disease occurs and becomes worse after operation. However, there is some justification in the procedure in that the tonsillectomized children had somewhat less carditis than the children who were not operated upon.

The whole subject of rheumatism can be summed up with this statement that, though statistically we cannot prove anything, there isn't anybody in this room who would not feel that there is some advantage in operating upon the rheumatic child.

We have been studying the effect of tonsil filtrates on the growth of streptococci in culture and in immunity studies. There was no difference in the effect on growth in streptococci in culture media or in animals. However, when we used the filtrate of the tonsillar streptococcus, we found that the tonsil of the child who had been free from infection did not act the same as the tonsil from the child who had had repeated infection.

Ultimately, chemical and dietary regulation may be more effective than surgery. I have made myself very unpopular in my community in trying to stop indiscriminate practice of tonsillectomy. Instead of 50 per cent, probably 10 or 15 per cent need the operation. I believe the otolaryngologists are in sympathy with this trend and will be glad to cooperate with us.

CHAIRMAN ABT.—One thought occurs to me in closing. We must study the individual.

In acute tonsillitis conservative treatment is needed. What are the criteria of chronic infection in the tonsil? We might find hemolytic streptococci or diphtheria

organisms in normal throats but do enlarged cervical glands, an increased leucocyte count or an increase of the polymorphonuclear cells mean anything to us?

DR. KAISER.—The four signs of tonsillar infection that can be observed are: the escape of pus from the crypts when pressure is applied to the tonsil; an enlarged tonsillar gland behind the angle of the jaw, varying in size and sometimes tender; a purplish red tinge limited to the anterior faucial pillar; and an abnormal preponderance of leucocytes in material from the tonsillar crypts, frequently accompanied by general hyperemia of the pharyngeal and palatal mucosa. The size of the tonsil is no criterion of the degree of infection within it.

DR. A. GRAEME MITCHELL (CINCINNATI, OHIO).—I am interested in the work of Jarvis, mentioned by Dr. Weston, and think something more should be said about it. We must admit there is something to the benefits of general diet and the upbuilding of health in relation to many conditions, including perhaps infections and infected tissue in the nasopharynx.

CHAIRMAN ABT.—First of all, we have learned that we are going to do everything that is reasonable to prevent hypertrophy and disease of the tonsils and of the pharyngeal lymphoid tissue; second, we are going to determine when the tonsils are diseased; and third, we are going to set a reasonable logical indication for leaving them alone. Then we are going to set a reasonable logical indication for their removal when they are diseased.

News and Notes

The following men have been certified by the **American Board of Pediatrics** since the last report:

Harold Abramson, New York, N. Y.
Donald Jerome Barnes, Detroit, Mich.
Harry S. Berman, Detroit, Mich.
Harry S. Bikoff, Brooklyn, N. Y.
Kenneth D. Blackfan, Boston, Mass.
Frank Otis Calaway, Houston, Texas
Moses H. Edelman, New York, N. Y.
John S. Fenby, Baltimore, Md.
Stanley Gibson, Chicago, Ill.
Roy M. Greenthal, Milwaukee, Wis.
Harry Mordecai Greenwald, Brooklyn, N. Y.
Arthur G. Helmick, Columbus, Ohio
Maurice Taggart Briggs, Lynn, Mass.
William J. Corcoran, Chicago, Ill.
Bradford French Dearing, San Francisco, Calif.
Helen G. Dennis, Portland, Ore.
Robert J. Dostal, Santa Monica, Calif.
Lucius Davis Hill, Jr., San Antonio, Texas
Edward James Lamb, Santa Barbara, Calif.
Jefferson Ralph Lemmon, Amarillo, Texas
W. McKim Marriott, St. Louis, Mo.
Robert George McAliley, Atlanta, Ga.
Samuel J. Levin, Detroit, Mich.
Ernest Langsdorff Noone, Drexel Hill, Pa.
Charles Sigmund Raue, Philadelphia, Pa.
Julian Leo Rogatz, New York, N. Y.
Abraham S. Small, Boston, Mass.
John Dawson Sturgeon, Jr., Uniontown, Pa.
Fritz B. Talbot, Boston, Mass.
Alfred S. Traisman, Chicago, Ill.
Joseph S. Wall, Washington, D. C.
Marshall Wallis, Houston, Texas
Joseph Ioor Waring, Charleston, S. C.
John H. McLeod, Washington, D. C.
Rudolph Duryea Moffett, New York, N. Y.
William A. Mulherin, Augusta, Ga.
H. T. Price, Pittsburgh, Pa.
Edwin F. Robb, Minneapolis, Minn.
Lewis Robbin, Newark, N. J.
Harold R. Roehm, Birmingham, Mich.
Councill Courtland Rudolph, St. Petersburg, Fla.
James Banister Stone, Richmond, Va.
John Garnett Young, Dallas, Texas

At the annual meeting of the **National Board of Medical Examiners** held at Atlantic City, it was voted to include pediatrics as one of the subjects in the examination for licensure by the National Board of Examiners. One and one-half hours in the written part of the Board's examination will be devoted to pediatrics.

At the meeting of the **Section on Pediatrics of the American Medical Association** Dr. Horton Casparis was elected section chairman for next year; Dr. H. L. Dwyer, vice chairman; and Dr. Ralph Tyson, secretary.

Dr. Isaac Abt, who for nearly twenty years has ably represented the pediatric section as their delegate to the House of Delegates of the American Medical Association, asked to be relieved of this duty. Dr. William Weston was elected delegate and Dr. A. Graeme Mitchell, alternate.

The **American Board of Pediatrics** announces the following examination dates:

August 8, Seattle, Wash.
October 10, Philadelphia, Pa.
November 10, St. Louis, Mo.

The Seattle examination, at the time of the meeting of Region IV of the Academy, is dependent upon a sufficient number of candidates for certification desiring examination at that time. The Philadelphia examination is scheduled for the day previous to the meeting of Region I, and the St. Louis examination at the time of the joint meeting of Regions II and III and the meeting of the Southern Medical Association.

The Academy Meeting in New York was saddened by the death of Dr. Orville Baldwin, of Columbus, Ohio. Dr. Baldwin accompanied by Mrs. Baldwin was in New York for the meeting. Friday night he had an attack of cardiac disease and died at the Waldorf early Sunday morning.

Dr. Charles A. Fife of Philadelphia, a Founder member of the Academy, died June 15 following an operation.

Dr. Mark Jampolis died May 19, following an abdominal operation. He had been well known in Chicago and the vicinity for many years as an upstanding and excellent, practical pediatrician. He was associated with Dr. Isaac A. Abt for several years and worked at Sarah Morris Hospital. Dr. Jampolis was fifty-four years old when he died.

Dr. Orville L. Baldwin, of Columbus, Ohio, died suddenly while attending the meeting of the Academy in New York. It is presumed that death was due to coronary thrombosis.

At a recent meeting of the Louisiana Pediatric Society held in New Orleans the following physicians were elected for the coming year: Dr. Charles James Bloom, of New Orleans, president; Dr. Ruth Aleman, of New Orleans, vice president; and Dr. J. A. Crawford, of Lake Charles, La., secretary and treasurer.

At the annual meeting of the Connecticut State Medical Society held in New Haven May 22 and 23, the following topics of pediatric interest were presented:

"Immunological Application of Placental Extracts," by Dr. Charles F. McKhann, Boston, Mass., and in coujunction with the Connecticut Society for Psychiatry, under the general topic of psychiatry for children, (1) "Twenty Years' Progress in Understanding Psychiatric Problems of Children" by Arthur A. Ruggles, M.D., Providence, R. I., President, National Committee for Mental Hygienes; (2) "The Best Current Practices in Psychiatry for Children," by George S. Stevens, M.D., Director, Division of Community Clinics, National Committee for Mental Hygiene.

Comments

WHEN the Academy of Pediatrics was founded a few years ago, a great many expressed the fear that it would be "just another medical society, just another meeting." The fifth annual meeting in New York on June 7 and 8 proved in many ways that these fears were groundless and that the purposes for which the Academy was founded and the ideas and ideals back of the original small group who were responsible for its birth are being realized. The four hundred fifteen who registered, nearly all of whom were members, testified to the interest of the membership and to the importance the Academy has assumed in their professional life. The meeting itself has developed into something quite different from the ordinary medical meeting for the presentation of papers; this is not the most important part of the Academy activities as is ordinarily the case with medical societies.

It is quite difficult to grasp and visualize the extent of the local, state, regional, and national activities of the Academy. We urge every member to read the committee reports which are published in this issue as it is only in this way that the earnest unselfish work, which literally hundreds of members of the Academy are engaged in throughout the year, can be understood, and it is this kind of activity that makes the Academy worth while. It is the development of activities of this character that has been responsible for the amazingly rapid growth of the Academy and for its having become in a few years an organization of vast importance and strength.

We wish in particular to call attention to the reports of two committees which during the present year have become extremely active. They are interesting further in that they illustrate the breadth of scope of Academy activities. One is the report of the Committee on Scientific Investigation, which proposes a "policy" for the Academy in regard to the utilization of research funds from commercial firms. This is a matter which within the last two or three years has led to a situation which has worried many because of its possible dangers in the field of pediatric investigation. We happen to know also that this committee is studying other important matters. The scope and work of such a committee so essentially national cannot be local. Their work, further, could never be done as individuals. In contrast to this the work of the Committee on Public Health Relations, if it is to be of any value, must be a combination of national, state, and local interests, and, as the excellent report shows, this has been adopted as the method of work. We mention these two reports as both of these committees have become exceedingly active during the past year. Many other groups are as active. The School Health, Hospital, and Educational committees, for example, show that their work is being continued.

It is necessary to think of the Academy in this way—not simply from the standpoint of the annual meeting and of the regional clinical meetings. Every member of the Academy is a part of this work and should take responsibility as well as pride in it.

The Journal of Pediatrics

CONTENTS FOR JULY, 1935

Original Communications

President's Address. Thomas B. Cooley, M.D., Detroit, Mich. ---- 1

Leucocytes in Infancy and Childhood. Katsuji Kato, M.D., Chicago, Ill. ---- 7

Erysipelas in Children Under Two Years of Age. Charles S. Culotta, M.D., New Haven, Conn. ---- 16

The Antirachitic Properties of Irradiated Evaporated Milk Fed to Normal Babies Under Home Conditions. Robert A. Strong, M.D., Emile F. Nacf, M.D., and Ina M. Harper, M.D., New Orleans, La. ---- 21

Intussusception Occurring in the Course of Paratyphoid Fever. Joseph Mignone, M.D., and Jay M. Arena, M.D., Durham, N. C. ---- 37

An Unusual Case of Glomerular Nephritis With Uremia. Roger H. Dennett, M.D., and Chester H. Myron, M.D., New York, N. Y. ---- 40

Spontaneous Hypoglycemia Due to Hyperinsulinism in a Child. Arthur M. Dannenberg, M.D., M. A. Bell, M.D., and Benjamin Gouley, M.D., Philadelphia, Pa. ---- 44

Infant Mortality in New York City One Hundred Years Ago. Charles Bolduan, M.D., and Louis Weiner, New York, N. Y. ---- 55

Is the Benefit of Lactic Acid Milk in Infant Feeding Due to the Bactericidal Action of the Lactic Acid? Kenneth B. Rothey, Durham, N. C. ---- 60

Bacteriologic Studies of Three Small Epidemics of Infectious Diarrhea in Children. Marion M. Johnston, Ph.D., and Mildred J. Kanke, Toronto, Ontario ---- 65

The Tonsil and Adenoid Problem. George Morrison Coates, M.D., and William Gordon, M.D., Philadelphia, Pa. ---- 70

Poisoning From the Bite of the Black Widow Spider (Latrodectus Mactans). E. H. Rohlfing, M.D., and B. M. Bullington, St. Louis, Mo. ---- 79

Vitamin A and Infection. Isaac F. Gittleman, M.D., and Alexander S. Wiener, M.D., Brooklyn, N. Y. ---- 81

Endocrine Studies in Infants and Children. II. Time of Onset of Teething, Walking, and Talking in Endocrine Disorders: Its Relationship to Intelligence in Endocrine and Nonendocrine Conditions. Murray B. Gordon, M.D., and Lawrence Kuskin, M.D., Brooklyn, N. Y. ---- 89

Critical Review

The Newborn. Barnet E. Bonar, M.D., Salt Lake City, Utah ---- 99

American Academy of Pediatrics

Proceedings: Fifth Annual Meeting of the American Academy of Pediatrics—Business Meeting ---- 118

Proceedings: Fourth Annual Meeting of the American Academy of Pediatrics—Round Table Discussion on the Tonsil Question ---- 137

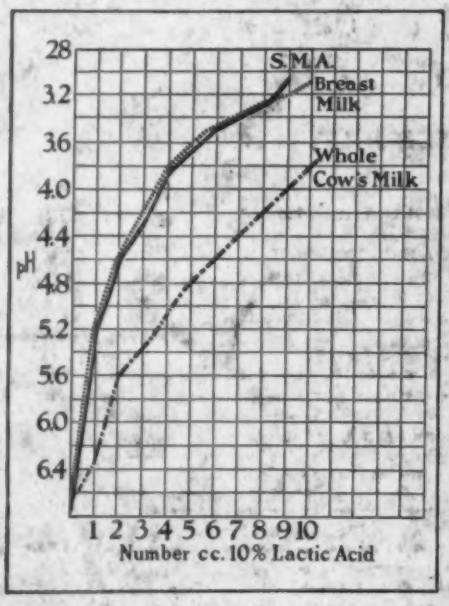
News and Notes

News and Notes ---- 154

Comments

Comments ---- 156

In BUFFER VALUE *too,* S.M.A. *resembles* BREAST MILK



This buffer chart shows the close similarity between the buffer value of S.M.A. and Breast Milk and the wide difference between cows' milk and Breast Milk. *This also explains why it is not necessary to add an acid to S. M. A.*

Breast Milk from the Normal Mother is the Ideal Food for the Human Infant. That is why S.M.A. is made to resemble breast milk in percentages of carbohydrate, protein, fat and total salts (ash) content, and why even the chemical and physical constants of the fat in S.M.A. are like those of breast milk fat.

This photograph shows the equipment for determining hydrogen ion concentration necessary to plot the Buffer Curve. The chemist drops a measured quantity of acid (from the tall tube in the right foreground) into solution to be tested (in the beaker below) and records the readings from the dial before him.

S.M.A. is a food for infants—derived from tuberculin tested cows' milk, the fat of which is replaced by animal and vegetable fats including biologically tested cod liver oil; with the addition of milk sugar and potassium chloride; altogether forming an antirachitic food. When diluted according to directions, it is *essentially similar to human milk* in percentages of protein, fat, carbohydrates and ash, in chemical constants of the fat and in physical properties.

S. M. A. CORPORATION « ◎ » CLEVELAND, OHIO